Dissertation on

AGGRESSIVE VERSUS EXPECTANT MANAGEMENT OF SEVERE PREECLAMPSIA REMOTE FROM TERM (28 – 32 WEEKS)

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M.D. DEGREE EXAMINATION

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CERTIFICATE

This is to certify that the dissertation titled **"Aggressive versus expectant management of severe preeclampsia remote from term (28 – 32 weeks)"** submitted by Dr. D. Anitha to the Faculty of Obstetrics and Gynaecology, Madras Medical College, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree (Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.

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This is submitted to the Tamilnadu Dr. MGR Medical University, Chennai in partial fulfillment of the rules and regulations for MD Degree Examination in Obstetrics and Gynaecology. This has not been previously submitted by me for the award of any degree or diploma from any university.

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INTRODUCTION

Preeclampsia complicating pregnancy is a common multisystem disorder characterized by blood pressure ≥ 140/90 mmHg after 20 weeks gestation with proteinuria ≥300mg/24 hours or ≥I +dipstick. Preeclampsia forms one of the deadly triad along with haemorrhage and infection that contributes greatly to maternal morbidity and mortality. It complicates 5-8% of the pregnancy. (ACOG 2002)[1]

Management of mild preeclampsia remote from term, the traditional approach adopted has been balancing the maternal and fetal outcome. Conversely management of severe preeclampsia remote from term has been delivering without delay regardless of fetal outcome.

With improved methods of monitoring and neonatal care facilities, several investigators has began to challenge the traditional view that women with severe preeclampsia need to be delivered expectantly.

Recent trend advocates expectant management in a selected group of women with preeclampsia remote from term with the aim of improving fetal outcome without compromising maternal safety. Gestational age at delivery, rather than the severity of maternal disease, is the primary determinant of perinatal outcome.

REVIEW OF LITERATURE

MINIMUM CRITERIA FOR DEFINING PREECLAMPSIA

(National high blood pressure education program 2000)[24]

1. New onset hypertension defined as blood pressure of 140/90 mmHg or higher after 20 weeks gestation.
2. New onset proteinuria defined as more than 300mg/24hrs or ≥1+ dipstick.

CLASSIFICATION OF PREECLAMPSIA

1. Mild preeclampsia
2. Severe preeclampsia

There is no category as moderate preeclampsia

ACOG CRITERIA FOR DIAGNOSIS OF SEVERE PREECLAMPSIA[1]

1. Systolic blood pressure of 160mmHg or higher or diastolic blood pressure of 110mmHg or higher on 2 occasions at least 6 hours apart while the patient is in bed rest after 20 weeks gestation.
2. Proteinuria of 5g or higher in a 24 hour urine sample or 3+ or greater on random urine sample collected at least 4 hours apart.
3. Oliguria of less than 500ml in 24 hours.
4. Pulmonary oedema.
5. Impaired liver function.
6. Thrombocytopenia.
7. Fetal growth restriction.
8. Cerebral or visual disturbance.
9. Epigastric or right upper quadrant pain.

BLOOD PRESSURE MEASUREMENT

Instrument

1. Conventional mercury sphygmomanometer:

   - gold standard for blood pressure measurement.

2. Electronic blood pressure monitor:

   - may under estimate the blood pressure.

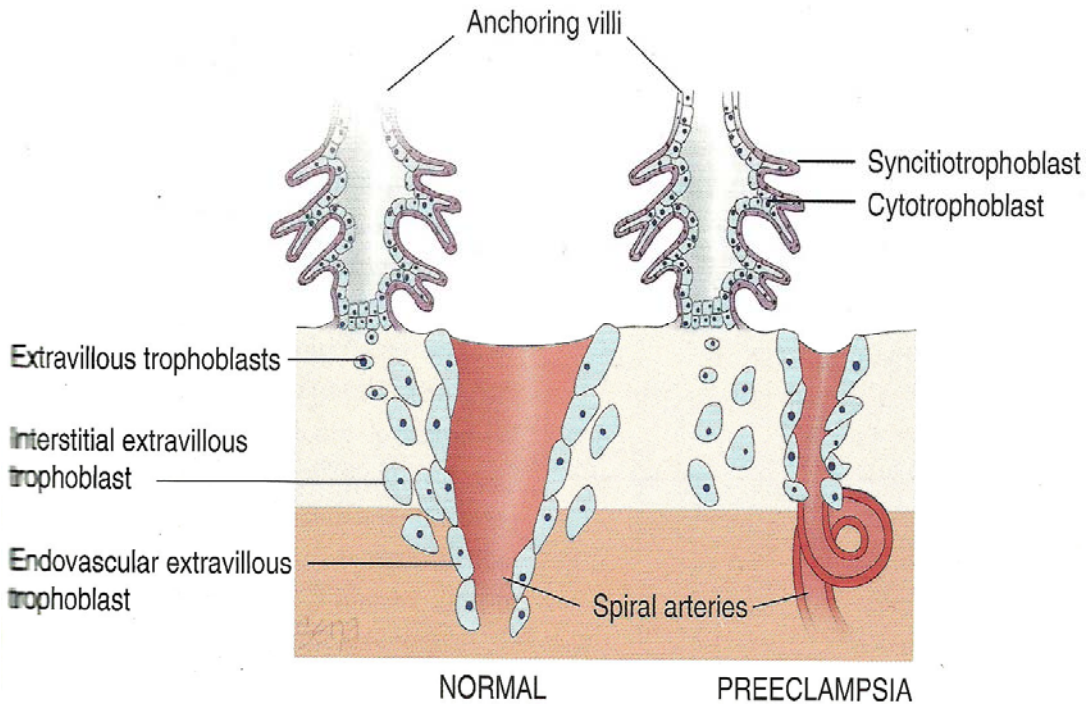
Technique

- Women should be relaxed and resting for at least half an hour before blood pressure measurement.
- Women should be seated or at 45° recline with the forearm horizontal and well supported and the arm at the level of heart with her feet supported or on the ground.
- Right arm should be used with a cuff of appropriate size.
- Korotkoff sound V should be used as a measure of diastolic blood pressure.
- K_5 is closer to the actual intra-arterial pressure, physiologically accurate, more reliably detected and reproducible.
- K_4 has limited reproducibility.
- K_4 /K_5 difference is smaller in hypertensive than in normotensive pregnant women.

THEORIES ABOUT ETIOPATHOGENESIS OF PREECLAMPSIA

1. ABNORMAL PLACENTATION

Failure of secondary wave of trophoblastic invasion into myometrial spiral arterioles results in reduced uteroplacental blood flow. The ensuing ischemia and hypoxia leads to aberrant expression of genes which encode for proinflammatory cytokines capable of eliciting endothelial dysfunction[5].



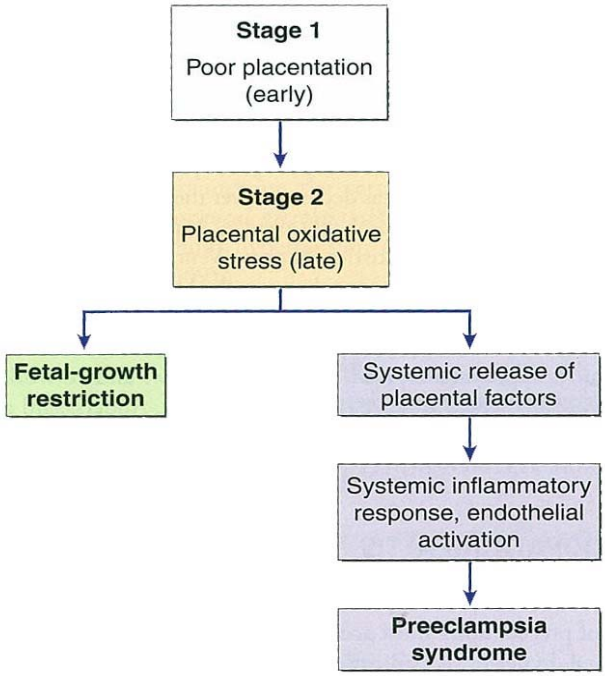
2. ENDOTHELIAL DYSFUNCTION

Endothelial dysfunction resulting in

a. Loss of vascular integrity is evidenced by elevated biomarkers of endothelial dysfunction such as plasma fibronectin and thrombomodulin[12].

b. Increased vascular reactivity results in generalised and intense vasospasm leads to reduced perfusion. This is due to increase in vasoconstrictors thromboxane and endothelin and increased sensitivity to angitensin II and decrease in vasodilators nitric oxide and prostacyclin[42].

c. Activation of coagulation cascade.



3. IMMUNOLOGICAL FACTOR

Immune maladaptation in preeclampsia.

a) Pathological lesions in placenta similar to acute graft rejection.

b) Lower level messenger RNA for HLA-G[8].

c) Th1/Th2 response with Th2 dominance. Cytokines like tumour necrosis factor-α, interleukin-2 and 6 mediate immune maladaptation[2].

d) Impaired production of blocking antibodies.

4. GENETIC PREDISPOSITION

Explained by both single gene model and polygenic inheritance.

a) Tendency for preeclampsia is inherited[6].

b) Women heterozygous for the angiotensin gene variant T235 had a higher incidence of preeclampsia[43].

c) Association between HLA DR4 and preeclampsia[17].

d) Inherited thrombophilia predispose women to preeclampsia

5. NUTRITIONAL FACTOR

Mac Gillivray viewed the evidence for the role of dietary deficiency in the pathology of preeclampsia

a) **Obesity:** C reactive protein is increased associated in turn with preeclampsia[46].

b) **Ascorbic acid:** Incidence of preeclampsia was doubled in women whose dietary intake of ascorbic acid was less than 85 mg[48].

c) **Calcium deficiency:** When concentration of calcium is low in extra cellular fluid amount of calcium entering the cell increases making vascular smooth muscle more sensitive to excitation.

6. OXIDATIVE STRESS

a) Preeclampsia may have its origin in a disturbed oxidative mechanism.

b) Abnormal levels of lipid peroxidise in preeclamptic women inhibits prostaglandin synthetase.

c) Risk of preeclampsia increased in women with increased oxidised low density lipoprotein and triglyceride[32].

PATHOPHYSIOLOGY OF PREECLAMPSIA

PRIMARY LEVEL

Changes that occur in placenta and placental vascular bed are

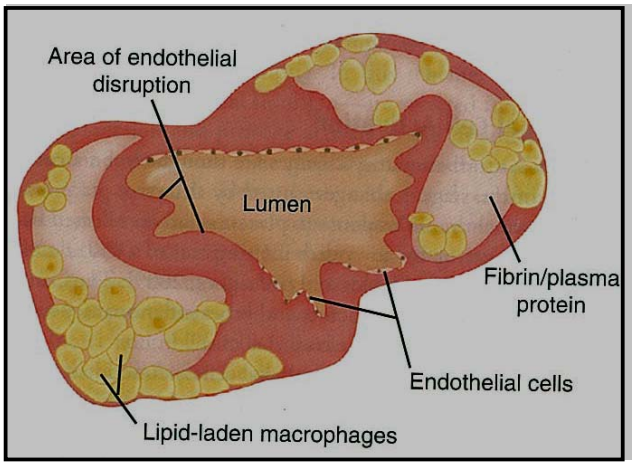
a) Relative lack of trophoblast infiltration into arterial wall during placentation.

b) Endothelial damage, insudation of plasma constituents into vessel wall, proliferation of myointimal cells and medial necrosis[10].

c) Acute atherosis.

d) Obstruction of spiral arteriolar lumen by atherosis may impair placental blood flow[29].

e) Magnitude of defective trophoblast infiltration of spiral arterioles correlated with the severity of hypertensive disorder[21].



SECONDARY LEVEL

I. Renal system

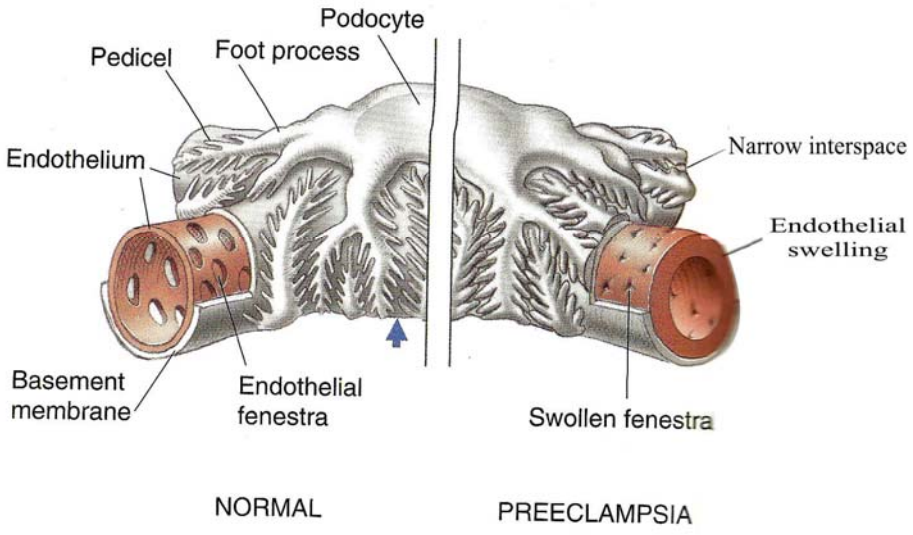
a) Renal Pathology - glomerular capillary endotheliosis[38]

b) Glomerular filtration rate and renal perfusion are decreased.

c) Hyperuricemia is due to reduced renal clearance of uric acid[7].

d) Proteinuria indicates advanced disease with poor prognosis.

e) Hypocalciuria is due to increased tubular resorption of calcium[39].



ii. Cardiovascular system

Cardiovascular dysfunction associated with preeclampsie are related to

a) Increased cardiac afterload caused by hypertension.

b) Decreased cardiac preload by hemoconcentration.

c) Endothelial activation with extravasation into the extracellular space.

1. Hemodynamic changes

a. Hyperdynamic circulation.

b. Increased peripheral resistance.

c. Decreased preload.

d. Decreased cardiac output.

e. Increased sensitivity to vasopressors.

2. Blood Volume Changes

a) Hypervolemia of pregnancy is severely curtailed.

b) Hemoconcentration due to generalised vasoconstriction and endothelial dysfunction with increased vascular permeability[37].

iii. Hematological changes

a) Microangiopathic hemolysis - caused by endothelial disruption with platelet aggregation and fibrin deposition, diagnosed by elevated levels of serum lactate dehydrogenase and peripheral smear schizocytosis[9].

b) Thrombocytopenia – due to platelet activation and aggregation[16].

c) Platelet activating factor increased.

d) Increased megakaryocytes and thrombopoetin.

e) Deficiency of any soluble coagulation factors very uncommon unless another event coexist[19].

f) Thrombophilia is associated with early onset preeclampsia.

g) Antithrombin III lowered.

iv. Hepatic changes

a) Elevation of liver enzymes in severe preeclampsia.

b) HELLP syndrome – hemolysis,elevated liver enzymes and low platelets[44].

c) Periportal hemorrhage in the periphery of liver.

d) Hepatic artery resistance increased evidenced by sonography[27].

Specimen – Liver damage in severe preeclampsia.

v. Endocrine changes

a) Renin, angiotensin II, aldosterone decrease to normal nonpregnant range[45].

b) Despite this women with preeclampsia avidly retain infused sodium.

c) Vasopressin level increase despite decrease in plasma osmolarity.

d) ANP levels increase in preeclamptic women.

vi. Fluid and electrolyte changes

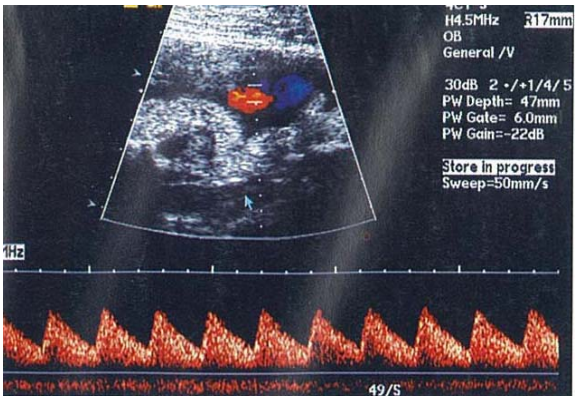
a) Extracellular fluid volume changes manifested as edema due to endothelial injury.

b) Electrolyte concentration do not differ much.

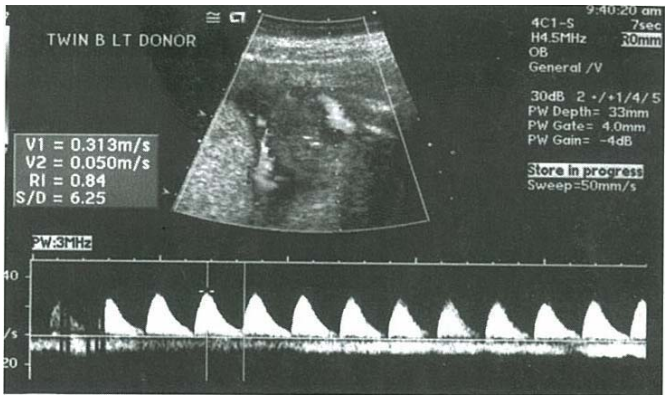
vii. Uteroplacental perfusion

a) Compromised uteroplacental perfusion from vasospasm is almost certainly a major culprit in the genesis of increased perinatal morbidity and mortality.

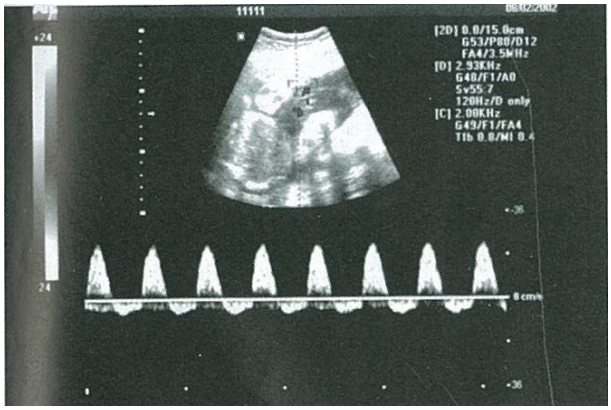
b) Doppler velocimetry measured higher impedance in peripheral than in central vessels – ring like distribution.



Normal umbilical artery velocimetry



Absent diastolic umbilical artery velocimetry

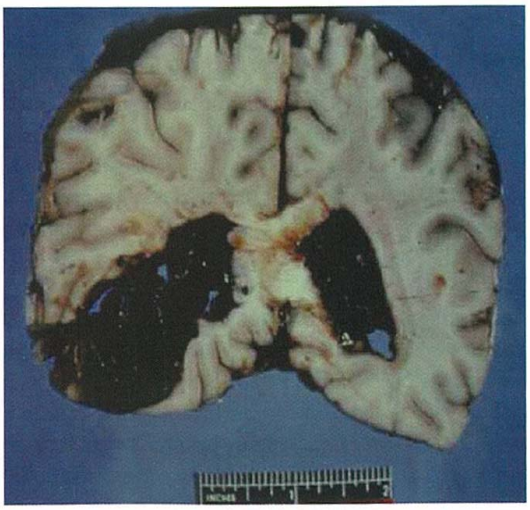


Reversal of umbilical artery velocimetry

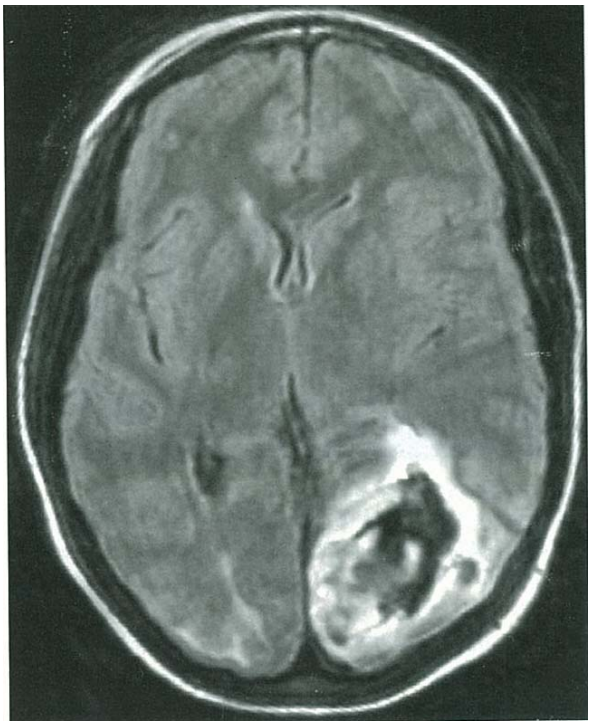
TERTIARY LEVEL

Tertiary systemic effects of preeclampsia are secondary to decompensation which can present as following

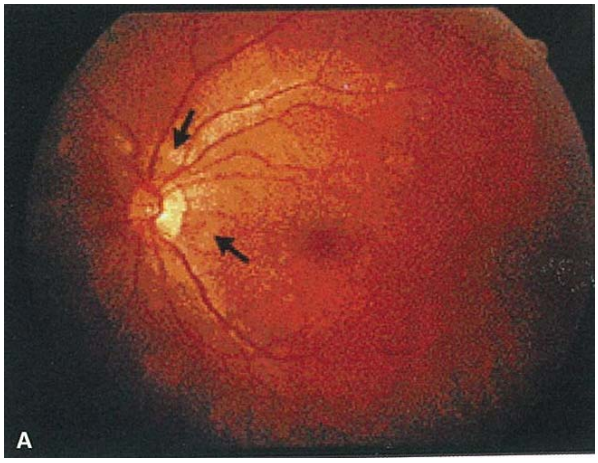
a. eclampsia
b. cerebral haemorrhage
c. pulmonary edema
d. ARDS
e. DIC
f. renal cortical necrosis
g. HELLP syndrome
h. retinal detachment
i. corneal edema
j. laryngeal edema
k. hepatic rupture



Specimen - intracranial hemorrhage



CT Brain - Intracerebral hemorrhage



Hypertensive retinopathy

FETAL AND NEONATAL EFFECTS

Preeclampsia is associated with increased perinatal morbidity and mortality.

a. Fetal growth restriction.

b. Intra uterine death.

c. Still birth.

d. Prematurity.

e. Respiratory distress syndrome.

f. Neonatal sepsis.

IUGR Baby

Preterm Baby

PREDISPOSING FACTORS OF PREECLAMPSIA

1. Age

a) Young primi < 20 years.

b) All patients > 30 years.

2. Parity and Paternity

a) Primi have higher incidence than multipara (11.9% vs 4.7%).

b) Protective effect of multipara is lost with change of partner[31].

c) Sperm exposure protects against preeclampsia. There is 2.4% increased risk for preeclampsia in contraceptive users.

d) Incidence of preeclampsia in nullipara 3.2%, multipara with changed paternity 3% compared with 1.9% multipara without change in paternity[40].

e) Donor insemination is associated with 2 fold risk for preeclampsia.

f) Oocyte donation is associated with increased risk for preeclampsia.

g) Men who fathered a preeclamptic women were nearly twice as likely to father a preeclamptic pregnancy in a different women.

3. Race

a) Arab, Muslims, Jews show an increased risk for preeclampsia.

b) Africo American ethnicity have an increased incidence.

4. Social status

a) Low socio economic status are reported to have increased incidence of preeclampsia.

b) Later studies showed that incidence was not different among, the five socio economic status.

5. Previous history of preeclampsia and interpregnancy interval

a) The risk of preeclampsia in subsequent pregnancies is higher when it is severe earlier.

b) Risk increases on increasing maternal age and interpregnancy interval.

c) 13.1% risk of preeclampsia in second pregnancy, risk of preeclampsia in second increases with maternal age 1.3 per 5 year of increase in age.

6. Family History

a) Severe preeclampsia and eclampsia have a familial tendency. 3 fold increase in preeclampsia and 4 fold increase of severe preeclampsia.

b) 26% incidence of preeclampsia in daughters.

c) Odds ratio 2.23 in full sisters, 1.6 in maternal half sisters and 1.8 in paternal half sisters.

7. Pregnancy Associated

a) Twin gestation: 4 fold increased risk of preeclampsia due to hyperplacentosis and relative placental ischaemia or immunological reaction to large placental mass.

b) Molar pregnancy: Confined to large rapidly growing moles in which the incidence of preeclampsia is 70%, with small slowly growing moles there is no increased incidence of preeclampsia.

c) Hydrops foetalis: Increased incidence of preeclampsia due to hyper placentosis.

d) Congenital malformation: In triploidy the risk of preeclampsia is 35% due to placentomegaly.

8. Urinary tract infection

This leads to increased production of inflammatory products, cytokines, free radicals and proteolytic enzymes causing endothelial dysfunction.

9. Obesity, insulin resistance, dyslipidemia

a) BMI is an independent risk factor for preeclampsia.

b) Adipocytes release tumour necrosis factor α aggravating cytokine mediated oxidative stress.

c) Insulin resistance / hyperinsulinemia are associated with increased sympathetic activity and increased tubular reabsorption of sodium.

d) Overt diabetes milletus 30% risk of preeclampsia especially when vascular changes are present. Odds risk increases by 20% per nmol/l increase in plasma glucose level.

10. Exogenous factors

a) Smoking: decrease the incidence of preeclampsia due to direct effect on placental function and reduction in HCG and estradiol[18].

b) Psychosocial strain: working women had 2.3 times risk of preeclampsia than non working women.

11. Underlying Disorders

a) Sickle cell trait women at significant risk of preeclampsia[20].

b) Women with familial thrombophilia especially those with combined defects and antithrombin deficiency have increased risk not only of preeclampsia but also fetal loss.

c) Hyper homocystenemia has 7times higher risk for preeclampsia[11].

d) Underlying renal disorder has 20% risk of preeclampsia.

MANAGEMENT OBJECTIVES IN SEVERE PREECLAMPSIA

There is no preventive therapy against preeclampsia at present. Even though calcium, aspirin, magnesium, fish oil have been tried. Severe preeclampsia is associated with maternal complication like HELLP, eclampsia, pulmonary edema, abruption, fetal complication like IUGR, IUD, preterm delivery .As termination of pregnancy remains the only cure the primary objective in the management of severe preeclampsia is to effect timely delivery in order to

- Prevent maternal morbidity and mortality.
- Improve the perinatal and neonatal outcome.

In all circumstance the wellbeing of mother is primary, in some cases delay seriously jeopardize the wellbeing of mother, fetus or both. Adopting expectant management in selected patients in a tertiary institution will improve the perinatal outcome without compromising maternal safety. Such an approach has been advocated by research workers in various part of world.

RANDOMISED CONTROLLED TRIAL AGGRESSIVE VERSUS EXPECTANT MANAGEMENT OF SEVERE PREECLAMPSIA REMOTE FROM TERM

Odendaal et al[25] at **1990** conducted the trial which included 58 women with severe preeclampsia between 28-34 weeks randomised to aggressive and expectant group. Results of the trial showed that in expectant management mean prolongation was 7.1 days, lower incidence of neonatal complications, less need for neonatal ventilation and not associated with increased maternal complications.

Sibai et al[36] at **1994** randomised 95 patients with severe preeclampsia between 28-32 weeks to aggressive and expectant management. Expectant group was managed with bed rest, antihypertensive drugs and intense maternal and fetal monitoring. In expectant group average latency period was 15.4 days, had higher birth weight, higher gestational age at delivery, lower admission to NICU, lower neonatal complications and similar incidence of abruption.

Visser et al[41] at **1995** managed 254 patients with severe preeclampsia between 20-32 weeks expectantly with the intention to prolong gestation. Outcome of the study was median prolongation of 14 days and perinatal mortality rate 20.5%. They concluded the study that

expectant management may delay delivery and enhance fetal maturity and doesn't appear to be associated with increased risk of maternal mortality and morbidity.

Hall DR et al[14] at **2000** did a 5 year prospective study to evaluate the perinatal outcome of early onset, severe preeclampsia in 340 patients. Clinical and biochemical monitoring of maternal status with careful blood pressure control. Fetal surveillance included six hourly heart rate monitoring, weekly Doppler and ultrasound evaluation of the fetus every two weeks. Results of the study was mean prolongation of 11 days, neonatal survival rate of 94%, NICU care in 40.7% of cases, median NICU stay was 6 days. 81.5% cases were delivered by caesarean section, most common indication being fetal distress. There was no maternal death. They concluded that expectant management of early onset, severe preeclampsia led to high perinatal and neonatal survival rates. However neonatal sepsis remains a cause for concern.

Shear RM et al[34] at **2005** conducted a retrospective study at Sainte-Justin hospital, Canada which included 155 women with severe preeclampsia < 34 weeks managed expectantly. Outcome of the study was mean latency period of 5.3 ± 5.2 days. Perinatal mortality rate of 3.9%. Gestational age was the strong predictor of perinatal outcome.

Sibai BM et al[35] at **2007** conducted a trial at university of Cincinnati, USA. 1677 women of gestational age between 24 and 34 weeks underwent expectant management. Study showed that expectant management in a suitable hospital is safe and it improves the neonatal outcome.

Sarasam DS et al[33] at **2008** evaluated expectant management in 35 women with early onset severe preeclampsia between 24-34 weeks. The study results were mean prolongation of 9.2 days, higher Apgar score at 1 minute, lower mean days of hospitalisation in the neonatal intensive care unit, with a lower incidence of neonatal and maternal complications. The study recommended expectant management in patients with severe preeclampsia remote from term, after proper selection of patients and careful monitoring.

Bombrys AE et al[4] at **2009** did a retrospective analysis of expectant management of severe preeclampsia in 66 patients at 27- 34 weeks gestation. All patients received corticosteroids. Median prolongation was 5 days. Birth weight <10% for gestational age in 27% neonates and < 5% gestational age in 8% neonates, supporting a role for such management in early onset severe preeclampsia. There was no eclampsia and 2 had transient renal insufficiency.

AGGRESSIVE VS EXPECTANT MANAGEMENT OF SEVERE PREECLAMPSIA REMOTE FROM TERM (28 – 32 WEEKS)

AIM OF STUDY

- To compare merits and demerits of aggressive vs expectant management of women with severe preeclampsia remote from term (28-32 weeks)
- To determine which is more beneficial by comparing perinatal and maternal outcome by statistical analysis

MATERIALS AND METHOD

- **Study Design** : Prospective study
- **Study period** : 2009-2010
- **Study Place** : Institute of obstetrics and gynaecology,Chennai.

SAMPLE

Group I Patient of severe preeclampsia remote from term (28-34 weeks) managed aggressively that is glucocorticoid treatment followed by delivery in 48 hours. All patients who delivered within 96 hours of admission were noted.

Group II Patients of same group treated expectantly i.e., glucocorticoid treatment followed by intensive maternal and fetal monitoring followed by delivery only for specific maternal and fetal indication beyond 96 hours

SAMPLE SIZE

100 patients in group I who delivered after 48 hours of steroid administration were compared with 100 patients of group II.

SELECTION CRITERIA

INCLUSION CRITERIA

1) Gestational age 28-32 weeks

2) Severe preeclampsia defined as

   i. Blood pressure ≥ 160/110 mmHg with proteinuria ≥ 2+

   ii. Blood pressure ≥ 150/100 mmHg with proteinuria ≥ 3+

   iii. Blood pressure ≥ 140/90 mmHg with proteiniuria, headache, oliguria.

EXCLUSION CRITERIA

1. Women with other medical complications

2. Rupture of membranes

3. Preterm labour

4. Multifetal gestation

5. Platelet count < 1,00,000 /mm^3 or HELLP syndrome

6. Eclampsia

7. Fetal congenital malformation

GUIDELINESS FOR EXPECTANT MANAGEMENT

1. All patients are observed in labour room for atleast 24 hours to determine their eligibility for expectant management
2. Magnesium sulfate for seizure prophylaxis for selected features
3. Steroids are given to improve fetal outcome
4. Anti-hypertensives are given to control blood pressure
5. Complete blood count, liver and renal function test were done

MATERNAL MONITORING

- Blood pressure every 4-6 hours
- Daily urine albumin
- Daily weight, gravidogram, urine-albumin
- Platelet count everyday
- LFT alternate day
- Serum uric acid biweekly
- Input - Output monitoring

- 24 hour urinary protein weekly
- Anti-hypertensive drugs to control blood pressure in the range of systole : 130-150 mmHg, diastole : 80-100 mmHg,
- Retinal changes.

FETAL

- Daily Fetal Movement count
- NST daily
- USG – for fetal growth weekly
- Doppler USG twice weekly

Then,

- Headache in preeclamptic women are treated with analgesic and bed rest.
- If headache persisted, blood pressure is uncontrolled, then decision is made for delivery and magnesium sulphate started.

WHAT TO EXPECT OF EXPECTANT MANGEMENT

At any time during the concerned period of prolonging pregnancy if contraindication to expectant management appears pregnancy is terminated either vaginally or abdominaly

INDICATIONS FOR TERMINATION

MATERNAL INDICATION

- Uncontrolled blood pressure ≥ 160/110 mmHg despite maximum dose of anti-hypertensives for 4 days
- Eclampsia
- Platelet count < 1,00,000 / mm^3
- SGOT/SGPT more than 2 times the upper limit of normal with epigastric pain/tenderness
- Pulmonary edema
- Compromised renal function
- Abruptio placenta
- Persistent severe headache or visual changes

FETAL INDICATION

- Repetitive late/variable deceleration
- AFI ≤ 5
- USG : absent (or) reversal of diastolic flow in umblical artery, EFW < 5^{th} centile

MODE OF TERMINATION

- LABOUR INDUCTION

  PGE_2 gel instillation for induction of labour - followed by oxytocin augmentation if needed.
- LSCS

GUIDELINES FOR AGGRESSIVE TREATMENT

- All patients are observed in labour room
- $Mgso_4$ regime to selected patients for seizure prophylaxis
- Steroids to improve fetal outcome followed by delivery in 48 hours

- Anti-hypertensive for blood pressure control, blood investigation done as like that of expectant management
- USG for fetal well being – then labour is induced by PGE_2gel followed by oxytocin augmentation if needed.

INTRAPARTUM MANAGEMENT

- Preeclamptic women are more prone to develop convulsion during labour than normotensive – hence if not intiated earlier $Mgso_4$ initiated during labour in selected cases
- Input/output monitoring
- If it is unripe LSCS to be considered because of high incidence of complications like abruption, fetal distress
- Epidural/general anaesthesia for LSCS
- Once cervix favourable augmentation given
- Local infiltration for vaginal delivery
- Fluid < 150ml/hr (if oliguria <10ml/hr fluid and $Mgso_4$ decreased accordingly)
- Anti-hypertensive treatment
- Goal : systolic Blood pressure 140-150 mmHg, diastolic Blood pressure 90-100 mmHg

POSTPARTUM MANAGEMENT

- Intensive monitoring done for 2-4 days – vitals, reflex, input output monitoring
- Blood Pressure control
- Prophylatic anticonvulsant not given
- Patients are seen at weekly interval until her Blood pressure is normal without medication
- If this change does't not occur by 6 weeks, workup for hypertension made.

OUTCOME

- Prolongation of pregnancy
- Perinatal outcome
- Maternal outcome were evaluated

Incidence in the study period:

Total number of deliveries:24,700

Mild preeclampsia:12%

Severe preeclampsia:1.3%

Eclampsia:0.4%

Table 1

Maternal age

Age (Years)	Aggressive (100)	Expectant (100)
16-19	16	13
20-24	33	36
25-29	29	27
30-34	19	20
35-40	03	04

Both groups were similarly matched with respect to their age group. 33% cases in aggressive group and 36% cases in expectant group belonged to 20-24 years of age.

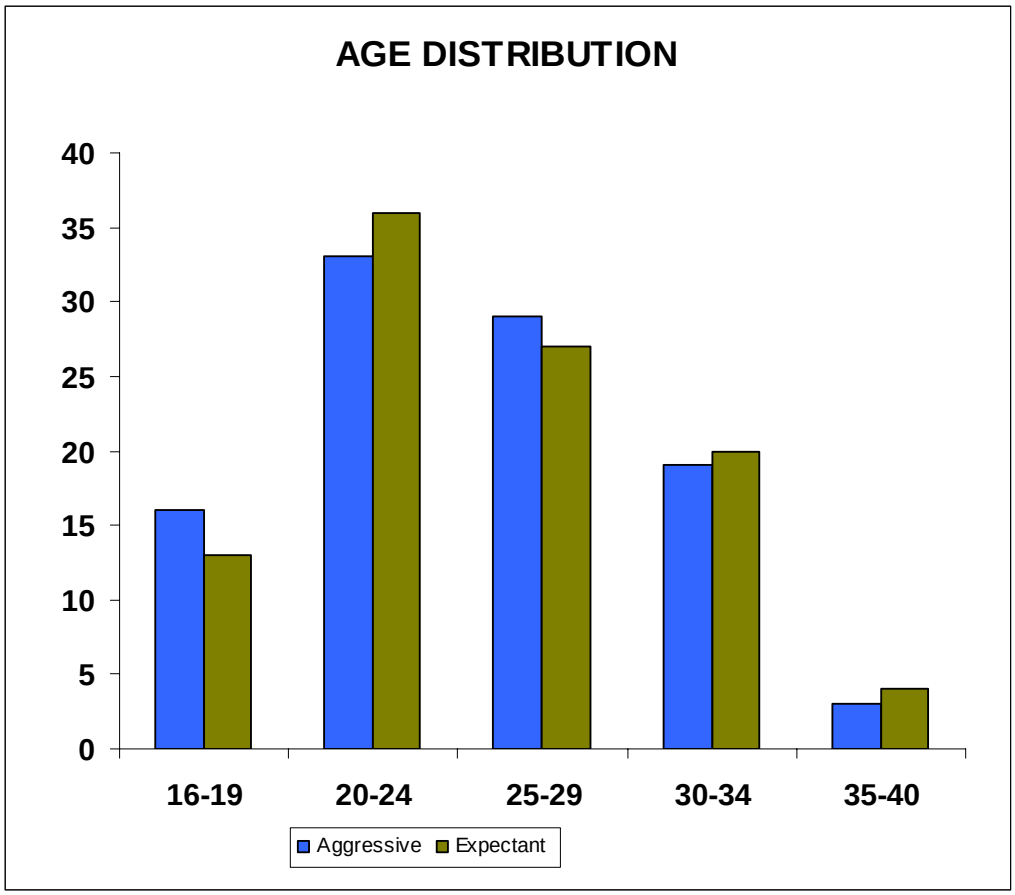

AGE DISTRIBUTION
40
35
30
25
20
15
10
5
0
16-19
20-24
25-29
30-34
35-40
Aggressive
Expectant

Table 2

Parity

Parity	Aggressive	Expectant
Primi	61	68
Second	24	28
Third	10	03
Fourth	04	01
Fifth	01	-

Both groups were similarly matched with respect to parity. Primi constituted 61% cases in aggressive group and 68% cases in expectant group.

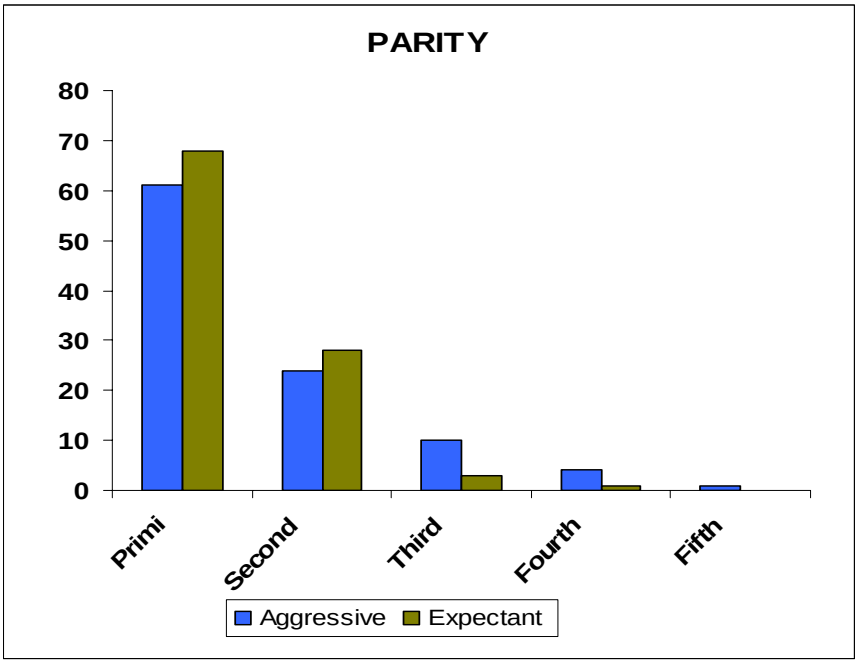


Table 3

Gestational Age on admission

GA weeks	Aggressive(100)	Expectant(100)
28-30	45	42
31-32	55	58

Both groups were similarly matched with respect to gestational age on admission. 55% cases in aggressive group and 58% cases in expectant group constituted 31-32 weeks gestational age,

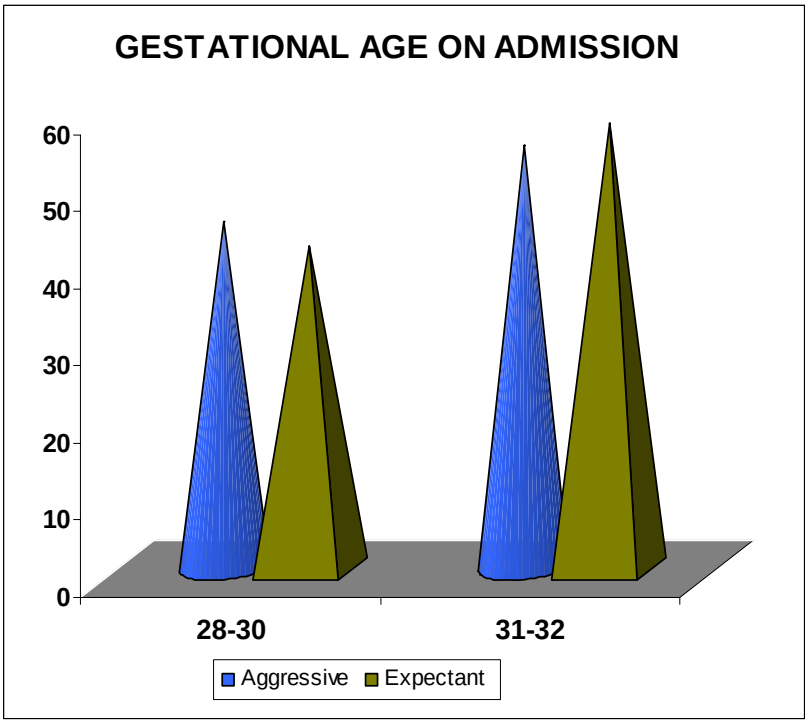


Table 4

Analysis of past obstetric history

Past H/o Preeclampsia	Aggressive (39)	Expectant (32)
Present	15	12
Absent	24	20

38.4% cases in aggressive group and 37.5% cases in expectant group had recurrent preeclampsia.

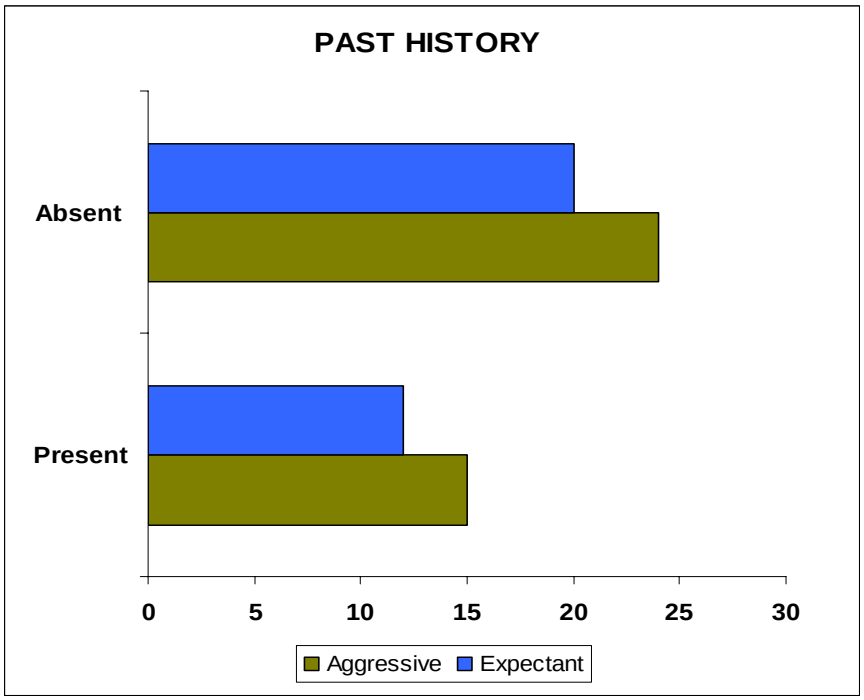


Table 5

Analysis of Family History

Family H/o	Aggressive (100)	Expectant (100)
Not known/no history	85	89
Mother preeclamptic	05	03
Sister preeclamptic	10	08

Only 15% cases in aggressive group and 11% cases in expectant group had family history of preeclampsia.

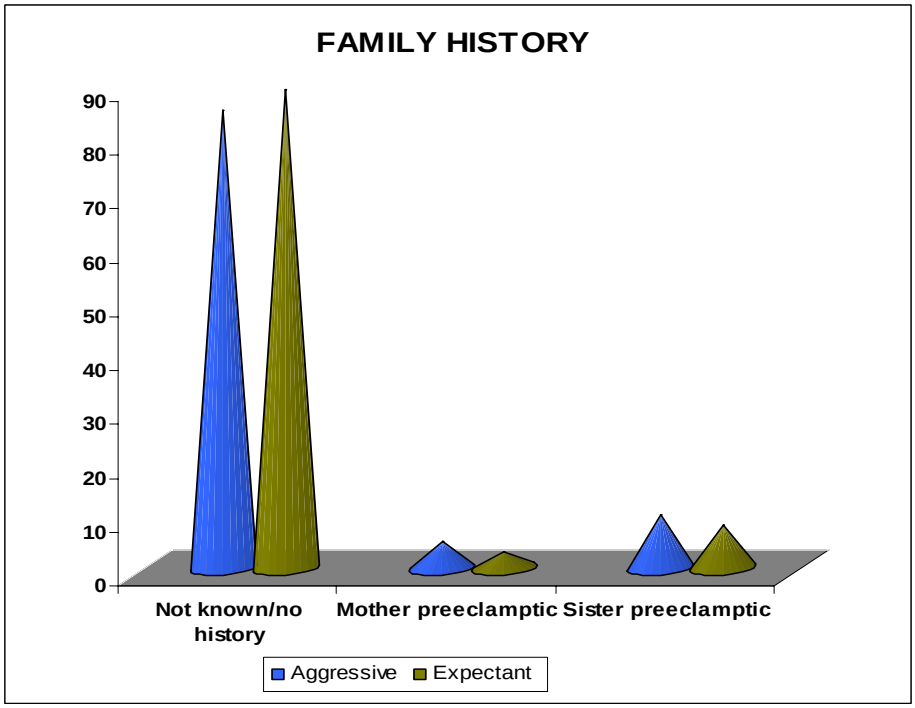


Table 6

Analysis of systolic blood pressure

Systolic BP on admission(mmHg)	Aggressive(100)	Expectant(100)
140-149	32	29
150-170	48	52
>170	20	19

Mean systolic blood pressure on admission was 157.42 mmHg in aggressive group and 157.38 mmHg in expectant group.

P value: 0.98 Statistically insignificant.

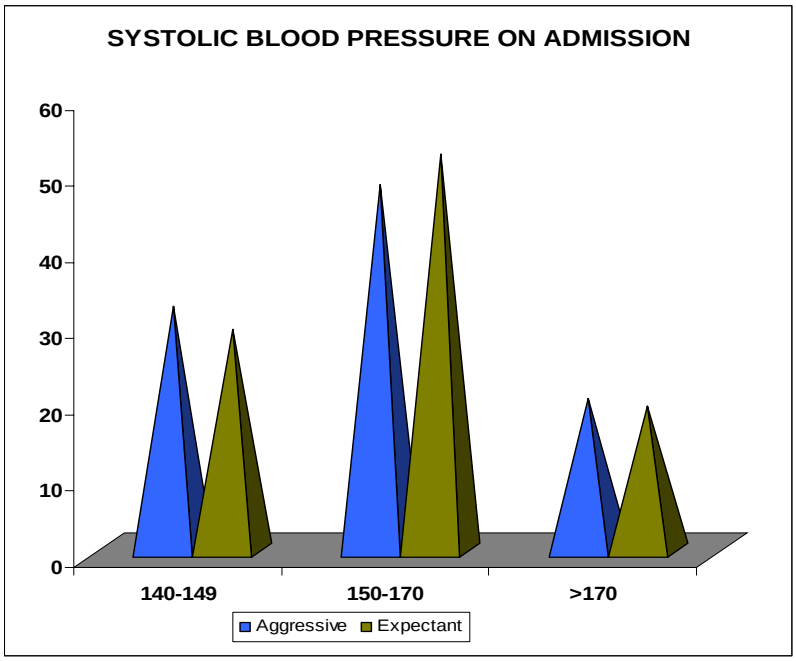


Table 7

Analysis of Diastolic Blood Pressure

Diastolic BP on admission(mmHg)	Aggressive (100)	Expectant (100)
90-100	39	28
101-110	35	43
111-120	23	25
>120	03	04

Mean diastolic blood pressure on admission was 104.67 mmHg in aggressive group and 105.73 mmHg in expectant group.

P value: 0.43 Statistically insignificant.

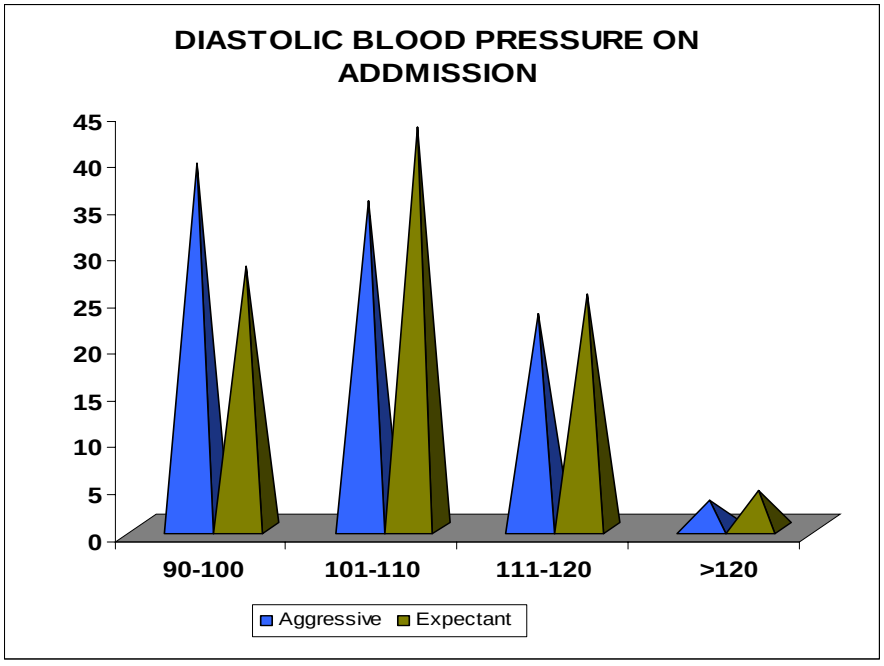


Table 8

Initiation of magnesium sulphate

$MgSO_4$	Aggressive (100)	Expectant (100)
Given	88	75
Not given	12	25

88% cases in aggressive group and 75% cases in expectant group were given magnesium sulphate.

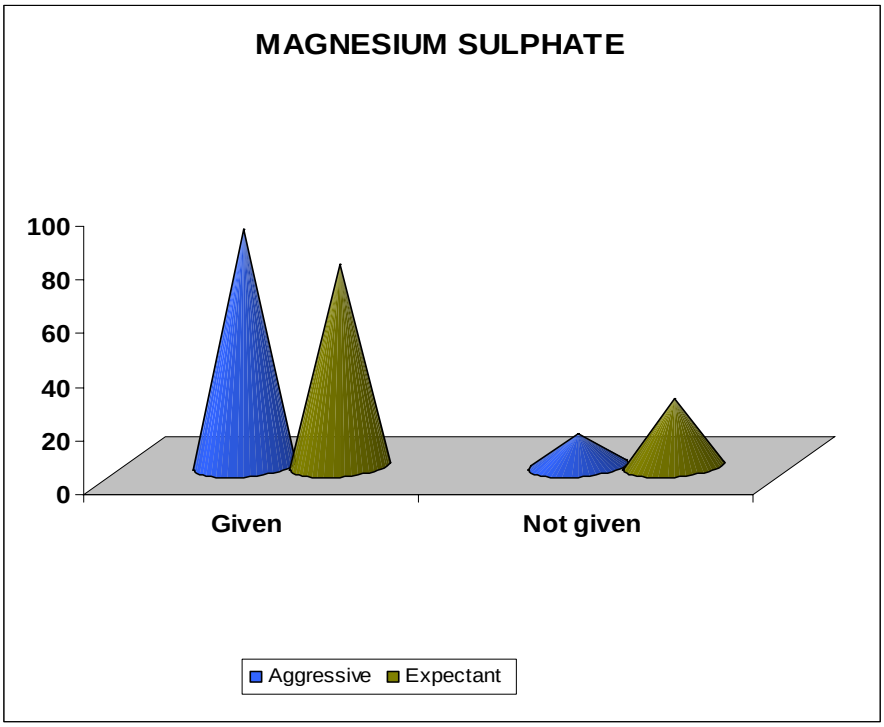


Table 9

Retinal changes

Fundus	Aggressive (100)	Expectant (100)
Normal	80	82
Grade I HTR	16	18
Grade II HTR	04	-

20% cases in aggressive group and 18% cases in expectant group had hypertensive retinopathy

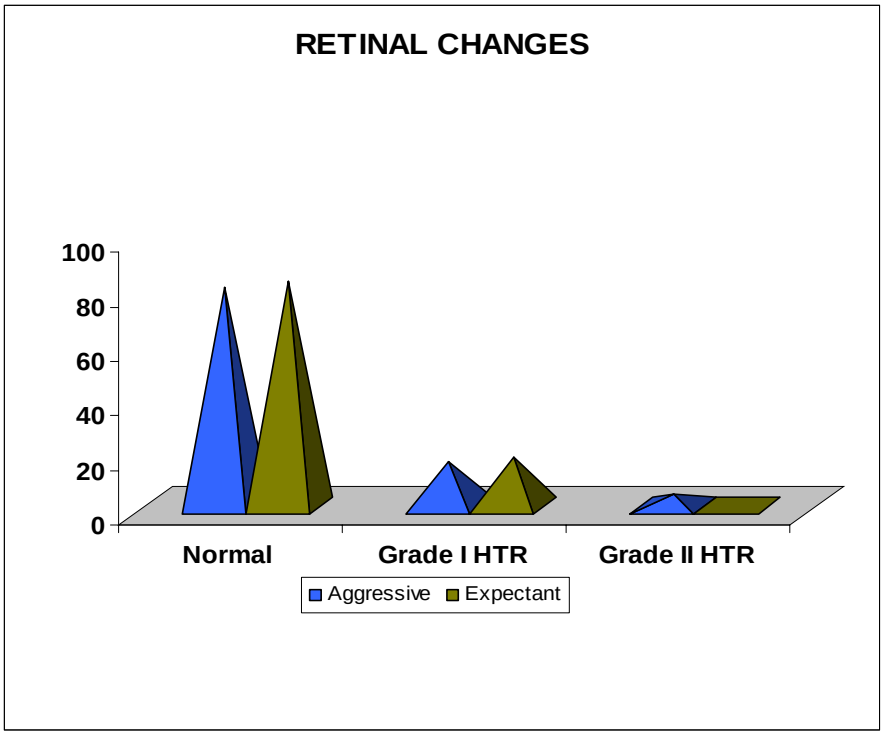


Table 10

Maternal Outcome

Complications	Aggressive				Expectant			
	AP	**IP**	**PP**	**Total**	**AP**	**IP**	**PP**	**Total**
Abruption	3	2	-	**5**	5	3	-	**8**
HELLP / DIC	2	-	-	**2**	1	2	-	**3**
Eclampsia	1	-	2	**3**	1	-	1	**2**
Renal failure	-	-	-	**-**	1	-	-	**1**
Pulmonary oedema	-	1	-	**1**	1	-	1	**2**
Cerebral oedema	-	-	-	**-**	-	-	1	**1**
Maternal death	-	-	-	**-**	-	-	-	**-**
Total	6	3	2	**11**	9	5	3	**17**

AP-Antepartum, IP-Intrapartum, PP-Postpartum.

11% major complications occurred in aggressive group while 17% complications in expectant group.

Though complications rate are slightly higher, they are well managed by anaesthetist and ICU indicating institutional supervision of expectant management.

It was found to be statistically insignificant.

Chi square value: 2.08

P value: 0.15

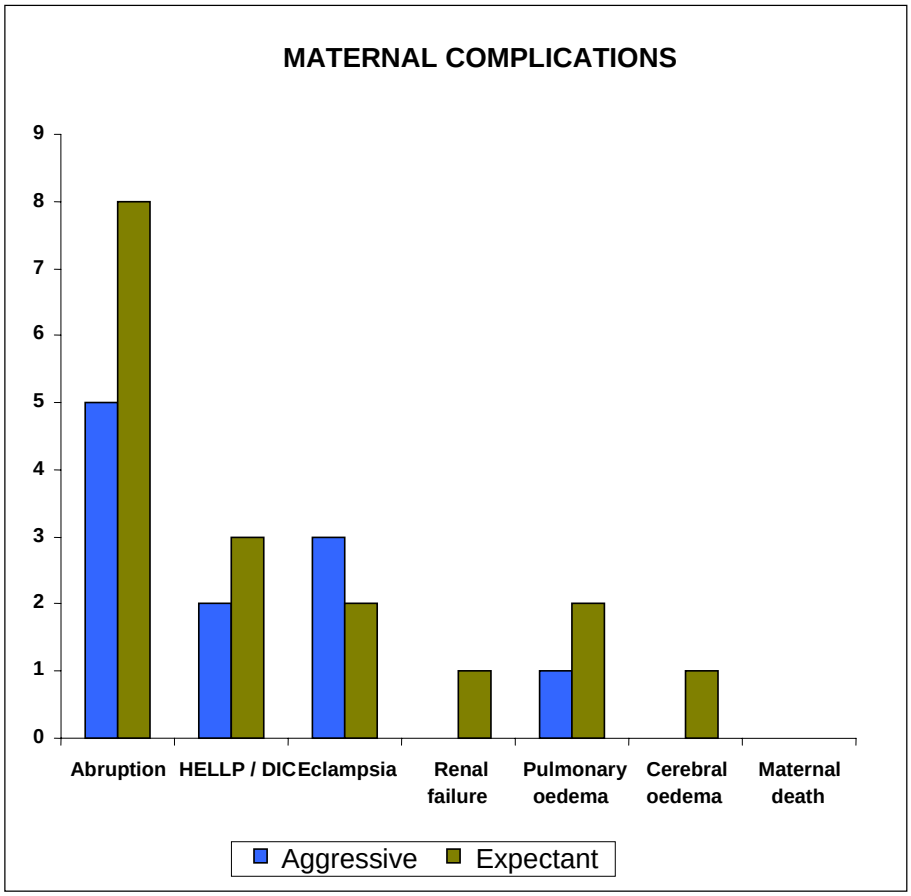

MATERNAL COMPLICATIONS
9
8
7
6
5
4
3
2
1
0
Abruption
HELLP / DIC
Eclampsia
Renal failure
Pulmonary oedema
Cerebral oedema
Maternal death
Aggressive
Expectant

Table 11

Indications for termination in expectant group

Indications	Expectant (100)
Maternal	66
Fetal	34

66% were terminated for maternal indications.

34% were terminated for fetal indications.

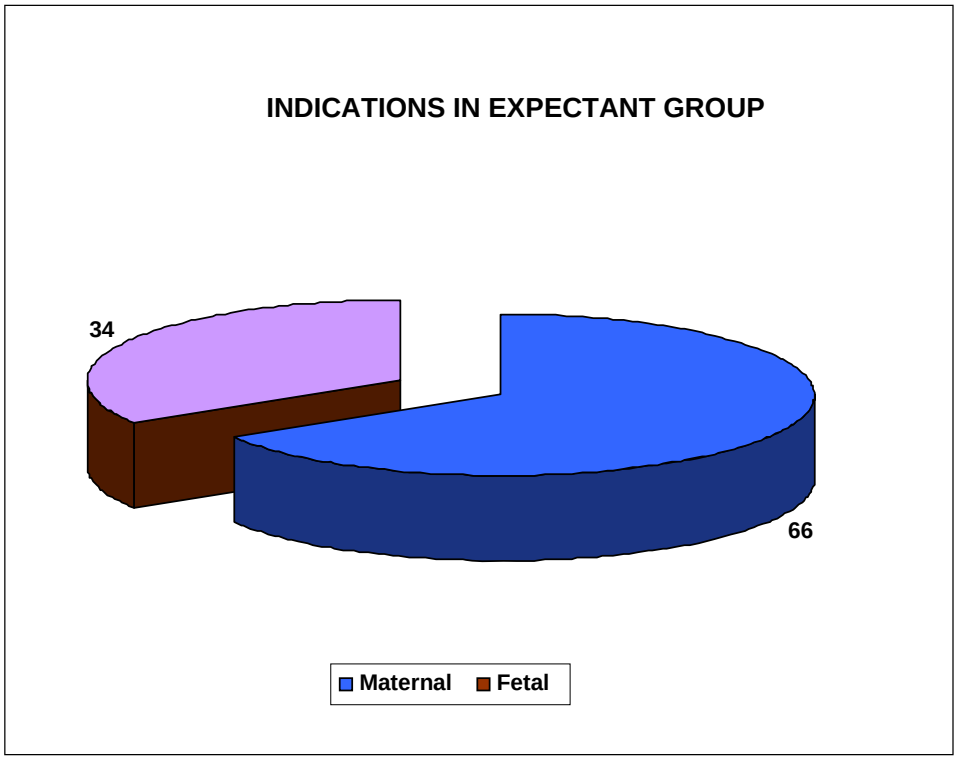


Table 12

Fetal indications for termination

Indication	Primi (68)	Multi (32)	Total (100)	Perinatal Loss
Severe oligohydraminos	08	03	11	05
Persistent late deceleration	06	02	08	04
IUGR <5th percentile	11	04	15	10
Total	**25**	**09**	**34**	**19**

Most common fetal indication being IUGR<5th percentile followed by severe oligohydramnios .

55% perinatal loss in cases terminated for fetal indications.

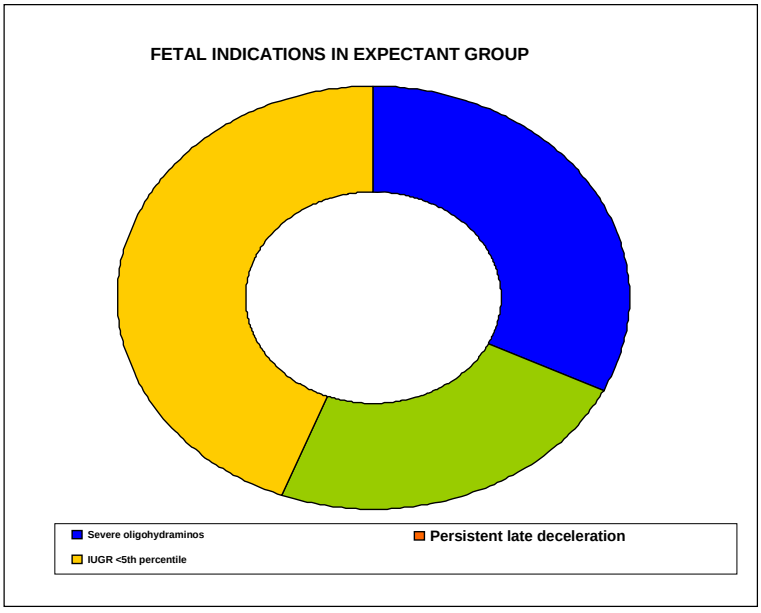


Table 13

Indications of termination in expectant group

Maternal indication

Indication	Primi (68)	Multi (32)	Total (100)	PN Loss
Imminent symptoms	22	10	32	09
Abruption	05	03	08	02
Uncontrolled BP	17	06	23	05
Compromised Renal function	-	01	01	-
Pulmonary edema	-	01	01	-
Eclampsia	01	-	01	-
HELLP	01	-	01	01
Total	**45**	**21**	**66**	**17**

PN- Perinatal

Most common indication being imminent symptoms followed by uncontrolled blood pressure.

25% perinatal loss in cases terminated for maternal indications.

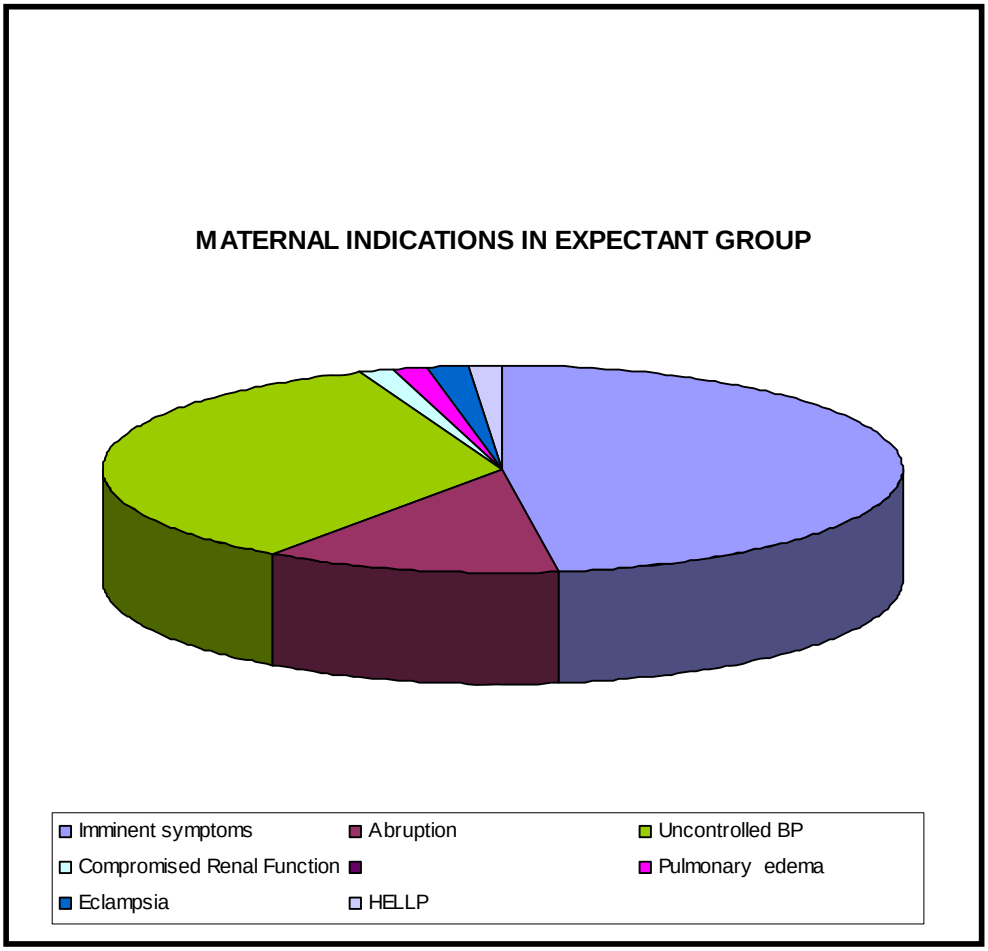

MATERNAL INDICATIONS IN EXPECTANT GROUP
Imminent symptoms
Abruption
Uncontrolled BP
Compromised Renal Function
Pulmonary edema
Eclampsia
HELLP

Table 14

Labour induction

Mode	Aggressive			Expectant		
	Primi (61)	Multi (39)	Total (100)	Primi (68)	Multi (32)	Total (100)
PG E_2 gel	52	33	75	53	19	72
Not induced due to maternal / fetal causes	09	16	25	15	13	28

75% cases were induced in aggressive group.

72% cases were induced in expectant group.

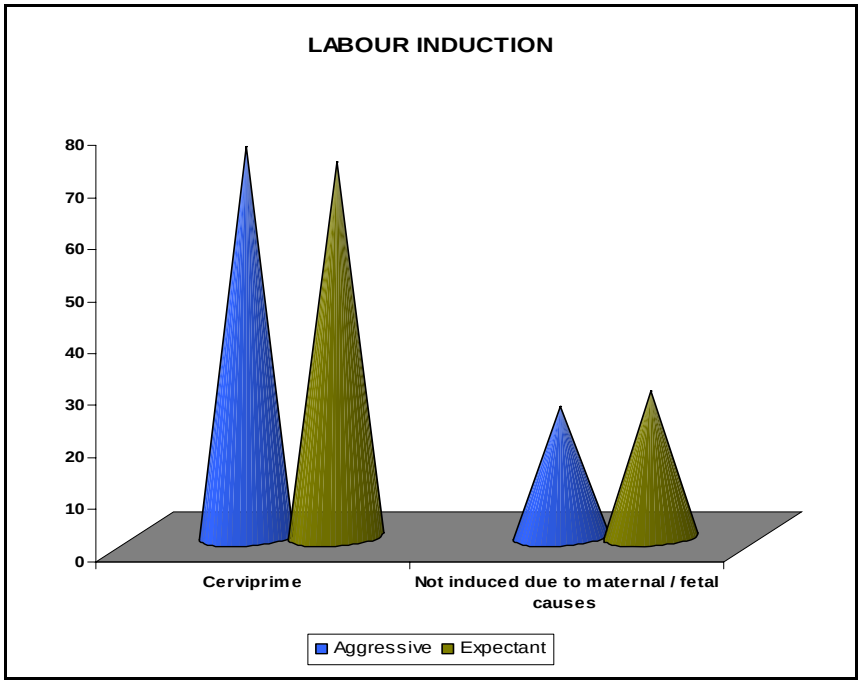


Table 15

Mode of delivery

Mode	Aggressive				Expectant			
	Primi (61)	**Multi (38)**	**Total (100)**	**PN loss (63)**	**Primi (68)**	**Multi (32)**	**Total (100)**	**PNLoss (36)**
Vaginal	48	31	**79**	**51**	42	20	**62**	**27**
LSCS	13	08	**21**	**12**	26	12	**38**	**09**

PN-Perinatal

- 21% LSCS in aggressive group whereas 38% LSCS in expectant group.
- As salvagability and fetal weight were lower in aggressive group, vaginal delivery preferred. Increased LSCS in expectant group was due to increased post ceaserean pregnancy.
- Perinatal loss in LSCS was 57% in aggressive group and 23.6% in expectant group.
- Chi square value : 1.41

  P = 0.23 Statistically insignificant.

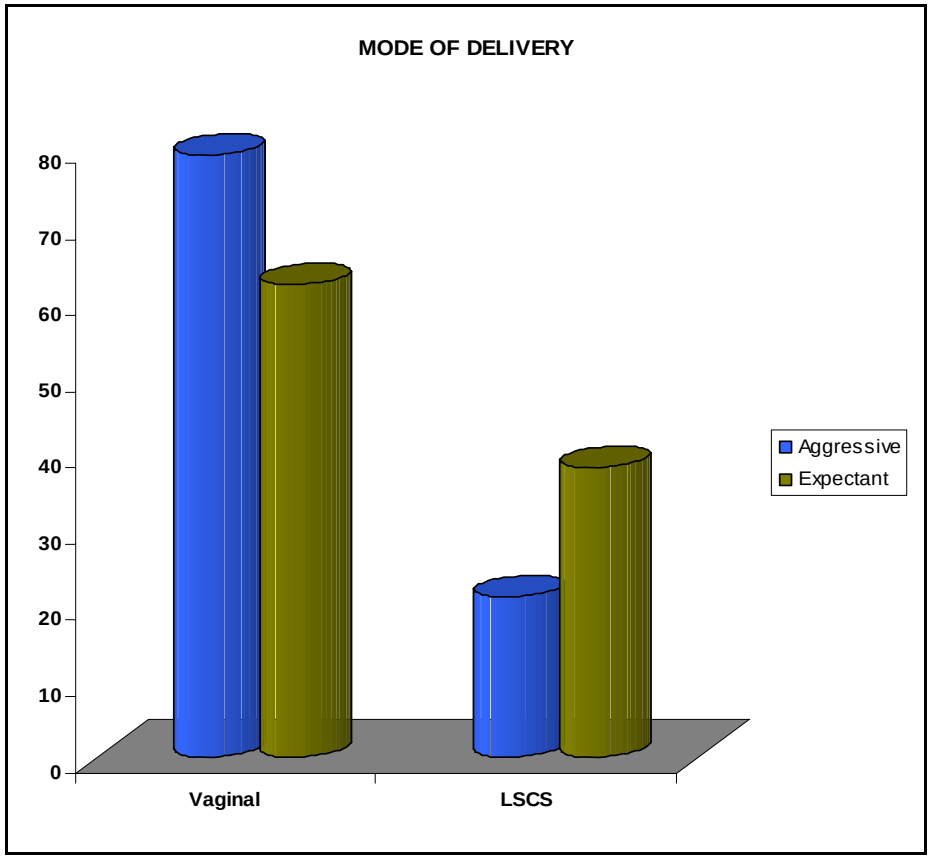

MODE OF DELIVERY
80
70
60
50
40
30
20
10
0
Vaginal
LSCS
Aggressive
Expectant

Table 16

Indication of LSCS in Aggressive group

Indication	Primi	Multi	Total	Perinatal Loss
Post LSCS	-	04	04	02
Non reassuring CTG	03	02	05	03
Malpresentation	01	-	01	01
Failure to progress unfavourable cervix	07	01	08	04
Abruption	02	01	03	02
Total	**13**	**08**	**21**	**12**

- Most common indication was Failure to progress/unfavourable cervix.
- Among 50% multipara the indication was post cesarean pregnancy.

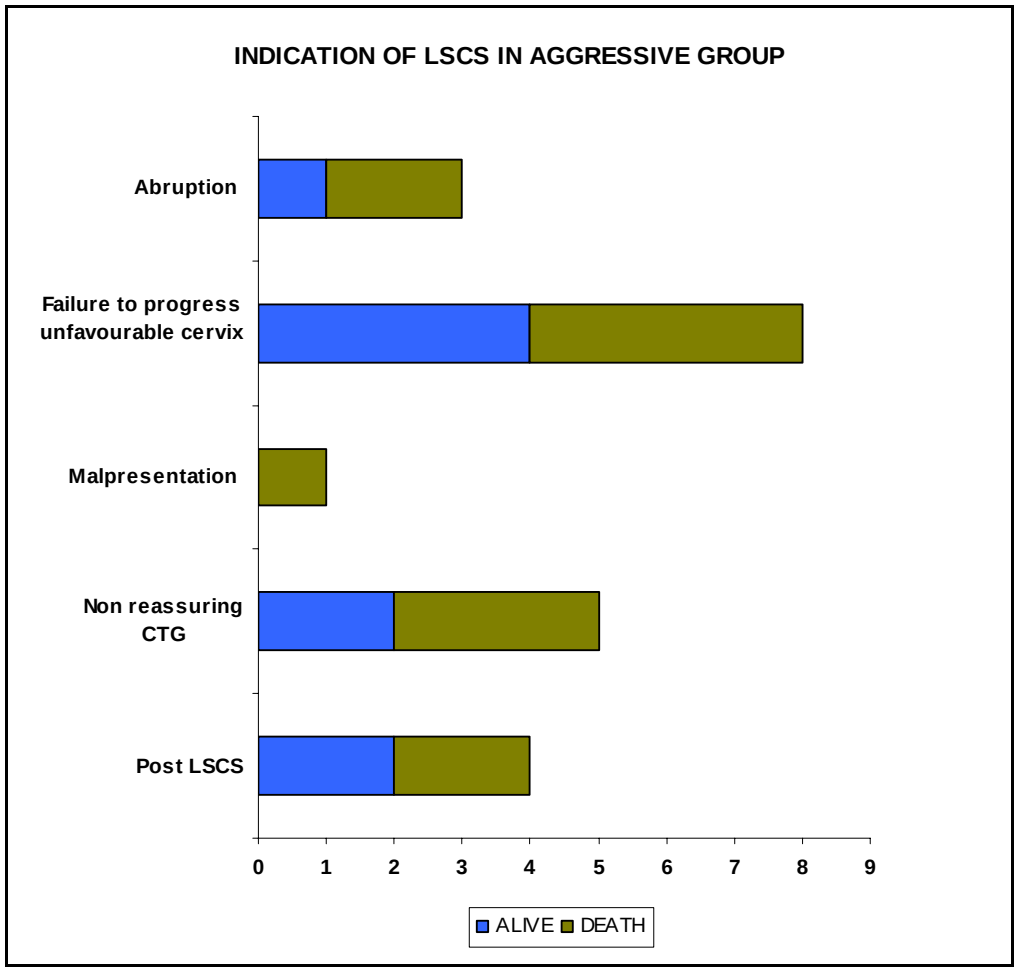

INDICATION OF LSCS IN AGGRESSIVE GROUP
Abruption
Failure to progress unfavourable cervix
Malpresentation
Non reassuring CTG
Post LSCS
0
1
2
3
4
5
6
7
8
9
ALIVE
DEATH

Table 17

Indication of LSCS in Expectant group

Indication	Primi	Multi	Total	Perinatal Loss
Post LSCS	_	07	07	01
Non reassuring CTG	10	02	12	03
Malpresentation	03	_	03	_
Failure to progress unfavourable cervix	11	02	13	03
Abruption	02	01	03	03
Total	**25**	**12**	**37**	**09**

- Most common indication was failure to progress/unfavourable cervix.
- Among 58.3% multipara the indication was post cesarean pregnancy.

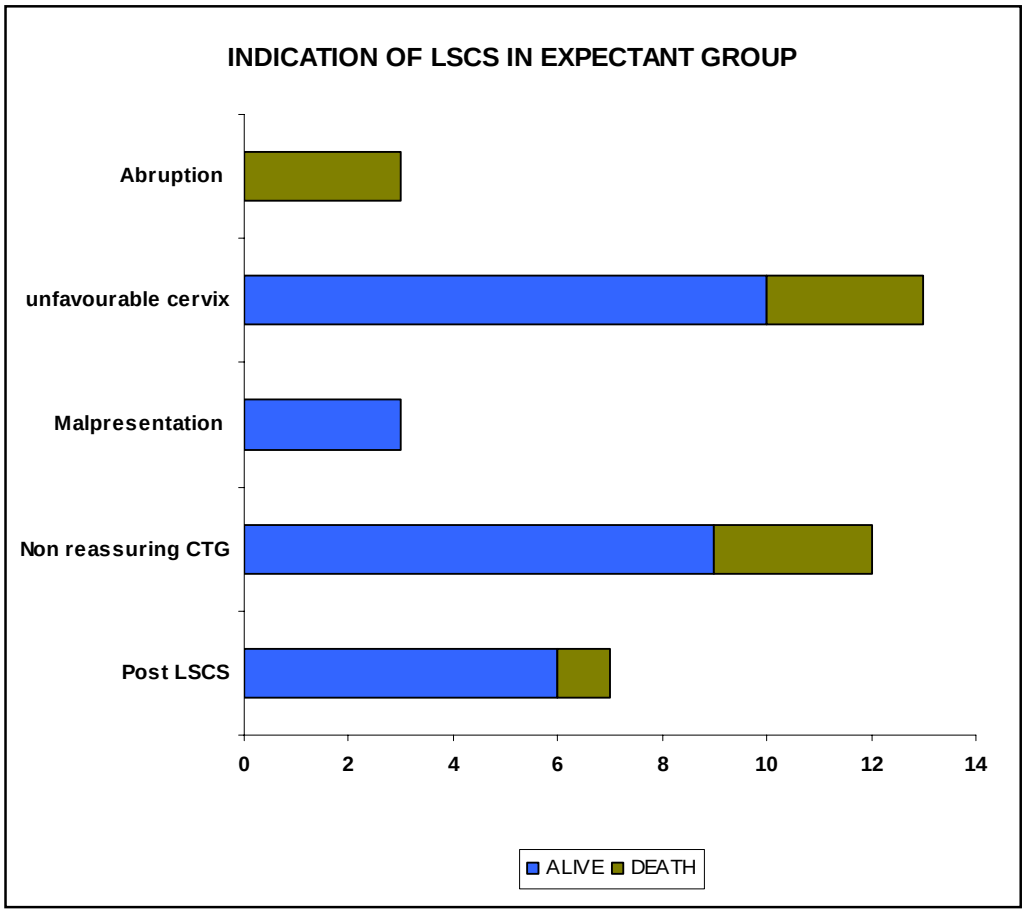

INDICATION OF LSCS IN EXPECTANT GROUP
Abruption
unfavourable cervix
Malpresentation
Non reassuring CTG
Post LSCS
0
2
4
6
8
10
12
14
ALIVE
DEATH

Table 18

Perinatal outcome in latency interval <96 hr

Latency interval (hours)	Cases	Perinatal Loss
< 24	31	23
24-48	27	19
49-72	53	34
73-96	47	27

- 58 patients who were delivered before 48 hours of steroid were not compared.
- 100 patients who were terminated after 48 hours of steroid were compared in our study.

Table 19

Prolongation of pregnancy in Expectant Group

Latency interval (Days)	**Cases**	**Perinatal Loss**
<5	23	16
5-8	40	13
9-12	29	05
13-20	06	02
>20	02	-
Total	**100**	**36**

Maximum prolongation was 23 days.

Median prolongation was 7 days.

Mean prolongation was 7.54 days

P value<0.001 Statistically significant.

As the latency interval increased perinatal loss decreased.

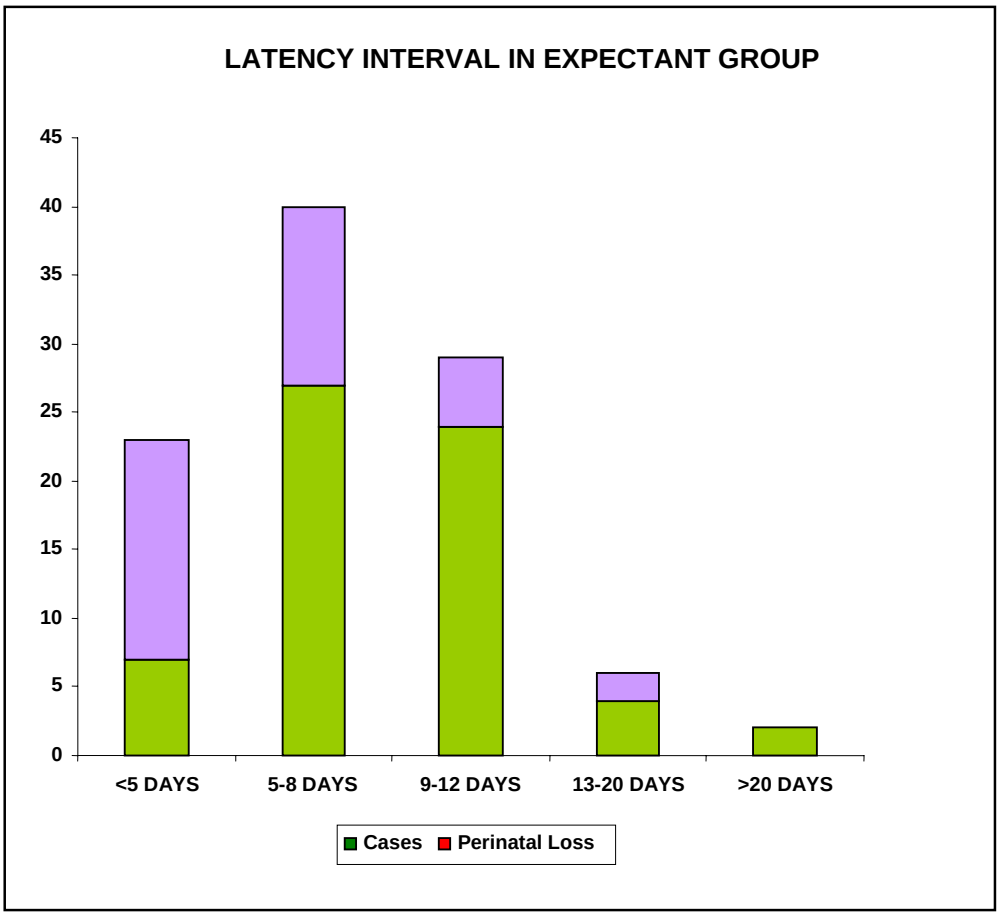

LATENCY INTERVAL IN EXPECTANT GROUP
45
40
35
30
25
20
15
10
5
0
<5 DAYS
5-8 DAYS
9-12 DAYS
13-20 DAYS
>20 DAYS
Cases
Perinatal Loss

Table 20

Fetal outcome

Fetal outcome	Aggressive			Expectant		
	Primi	**Multi**	**Total**	**Primi**	**Multi**	**Total**
Total Birth	61	39	**100**	68	32	**100**
Live Birth	45	31	**76**	60	27	**87**
Still Birth	16	8	**24**	10	3	**13**
Neonatal Death	28	11	**39**	17	6	**23**
Perinatal Death	44	19	**63**	27	9	**36**

Perinatal loss includes both still birth and early neonatal death.

Perinatal loss in aggressive group: 63%

Perinatal loss in expectant group: 36%

For live born

Chi square value: 4.01 Degree of freedom: 1

P value <0.05 Statistically significant.

For perinatal loss

Chi square value: 14.58 Degree of freedom: 1

P value<0.001 Statistically very high significance.

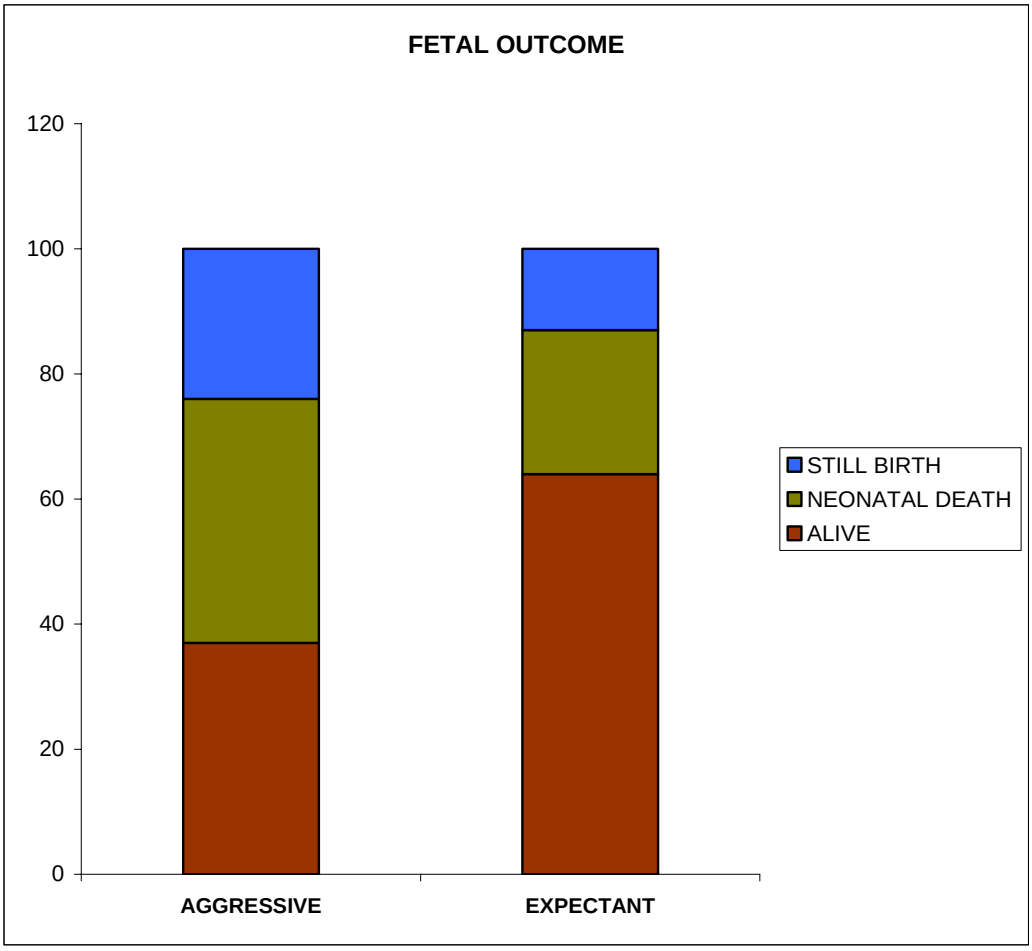

FETAL OUTCOME
120
100
80
60
40
20
0
AGGRESSIVE
EXPECTANT
STILL BIRTH
NEONATAL DEATH
ALIVE

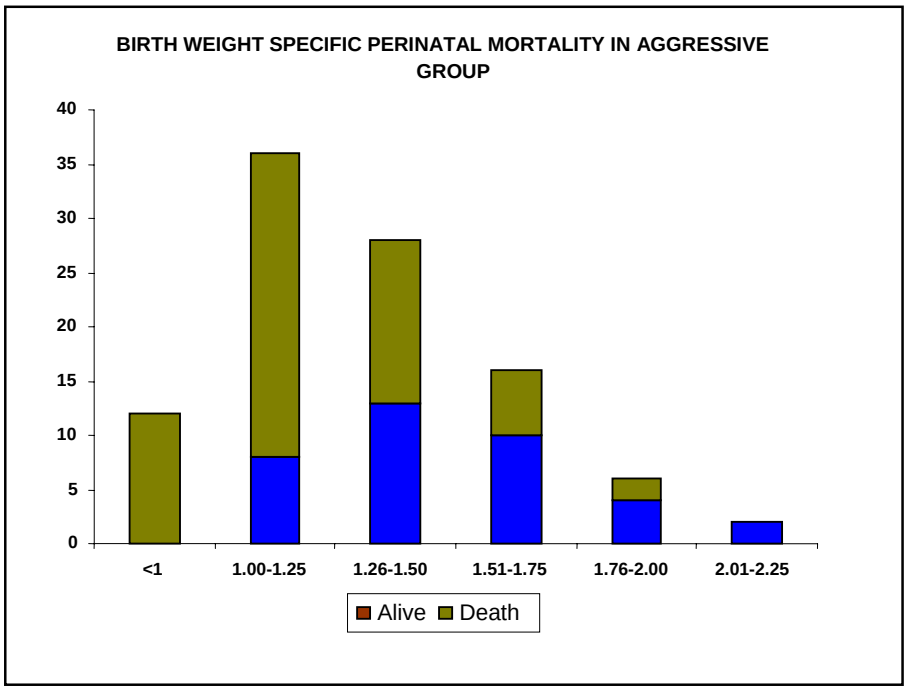
Table 21

Birth weight specific death

Birth weight (Kg)	Aggressive		Expectant	
	Born	**Death**	**Born**	**Death**
<1	12	12	06	06
1.00-1.25	36	28	16	12
1.26-1.50	28	15	16	08
1.51-1.75	16	06	34	08
1.76-2.00	06	02	16	02
2.01-2.25	02	_	12	_
Total	**100**	**63**	**100**	**36**

Mean birth weight was 1.33 kg in aggressive group and 1.61 kg in expectant group.

Pvalue <0.001 Statistically significant.


BIRTH WEIGHT SPECIFIC PERINATAL MORTALITY IN AGGRESSIVE GROUP
40
35
30
25
20
15
10
5
0
<1
1.00-1.25
1.26-1.50
1.51-1.75
1.76-2.00
2.01-2.25
Alive
Death

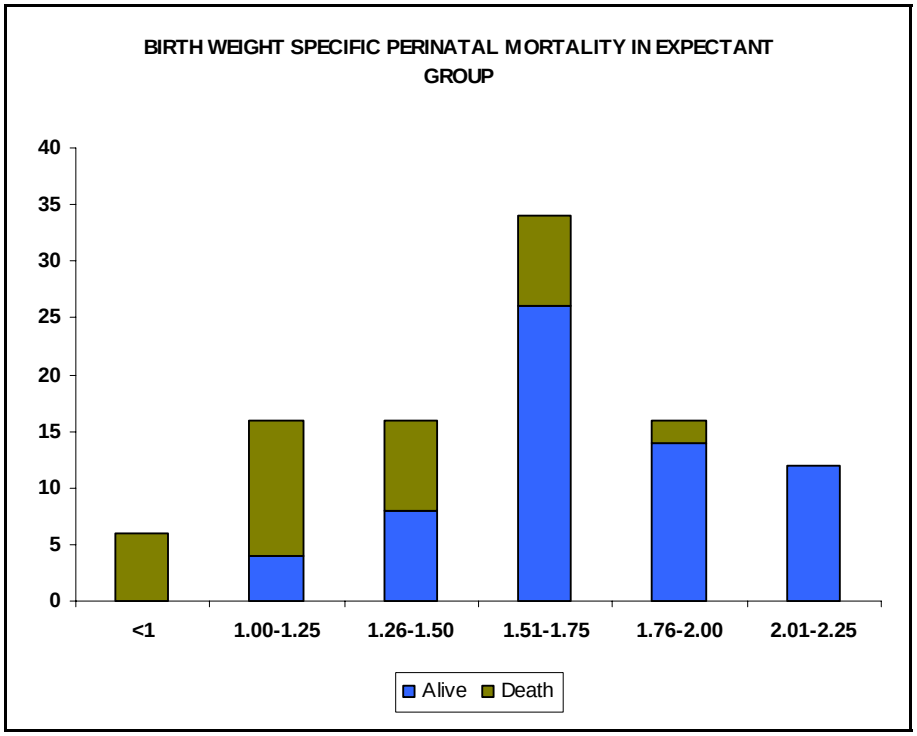

BIRTH WEIGHT SPECIFIC PERINATAL MORTALITY IN EXPECTANT GROUP
40
35
30
25
20
15
10
5
0
<1
1.00-1.25
1.26-1.50
1.51-1.75
1.76-2.00
2.01-2.25
Alive
Death

Table 22

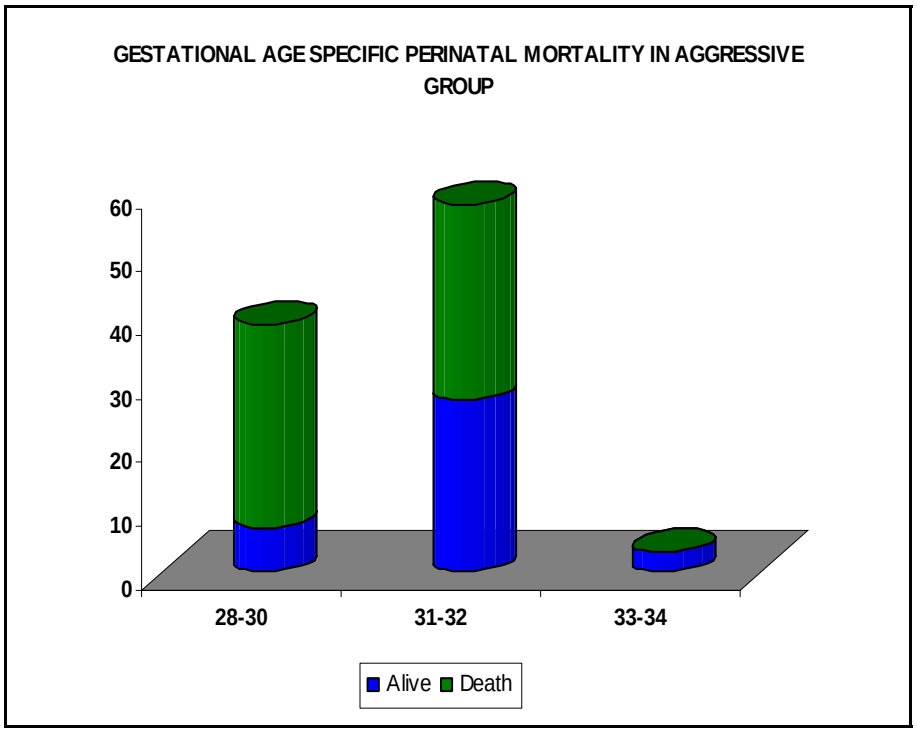
Gestational Age specific perinatal mortality

Gestation Age (Weeks)	Aggressive		Expectant	
	Born	**Death**	**Born**	**Death**
28-30	39	32	22	14
31-32	58	31	32	18
33-34	03	_	45	04
Total	**100**	**63**	**100**	**36**

Mean gestational age at delivery was 30.55 weeks in aggressive group and 31.64 weeks in expectant group.

Chi square value: 8.94

P value< 0.01 Statistically significant.


GESTATIONAL AGE SPECIFIC PERINATAL MORTALITY IN AGGRESSIVE GROUP
60
50
40
30
20
10
0
28-30
31-32
33-34
Alive
Death

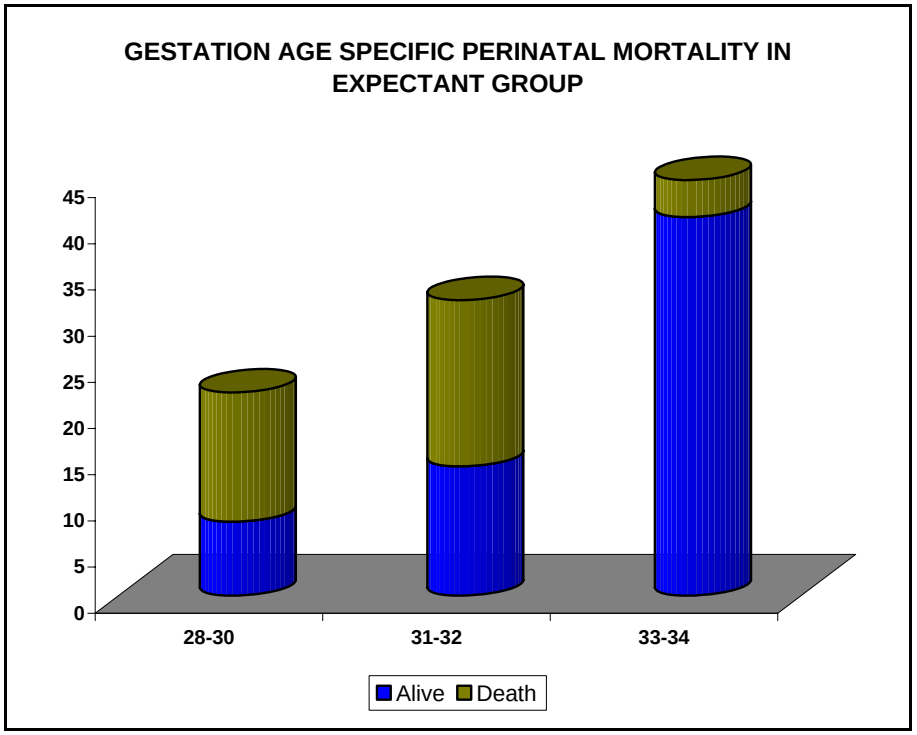

GESTATION AGE SPECIFIC PERINATAL MORTALITY IN EXPECTANT GROUP
45
40
35
30
25
20
15
10
5
0
28-30
31-32
33-34
Alive
Death

Table 23

Neonatal hospitalisation

Live Babies		**Aggressive (76)**	**Expectant (87)**
Admission to NICU		**76**	**87**
Neonatal Death		39	23
Survival hospital stay (Days)	< 5	03	20
	5-10	10	21
	11-15	09	14
	> 15	15	09
Total survival		**37**	**64**
Survival rate		**48.6%**	**73.5%**

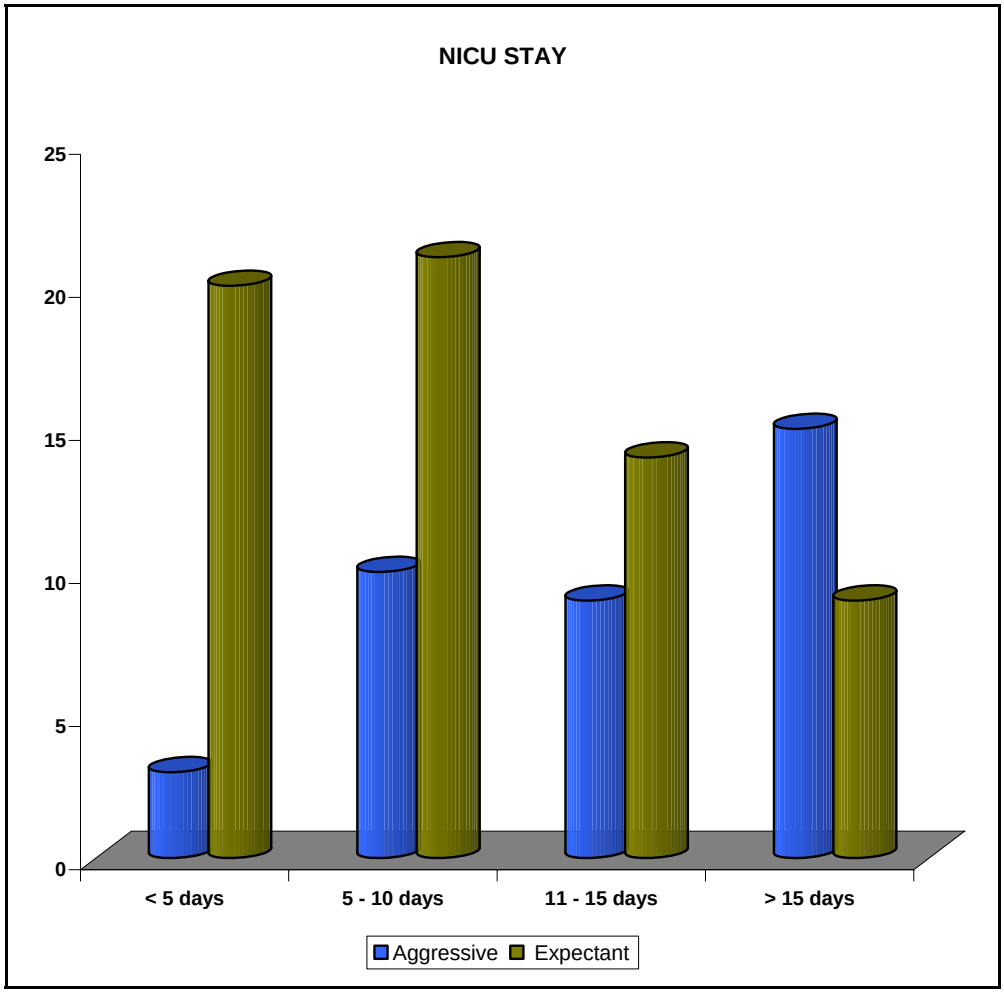
Mean hospital stay was 9.6 days in expectant group whereas in aggressive group it was 13.4 days.

Total survival was 37 babies in aggressive group.

Total survival was 64 babies in expectant group.

Chi square value:14.58

P value<0.001 Statistically significant.


NICU STAY
25
20
15
10
5
0
< 5 days
5 - 10 days
11 - 15 days
> 15 days
Aggressive
Expectant

DISCUSSION

- Incidence of preeclampsia is 5 – 8% according to ACOG 2002.
- Incidence of preeclampsia in IOG is 12%
- Incidence of severe preeclampsia in IOG is 1.3%
- There were 258 cases of severe preeclampsia remote from term 28 – 32 weeks in our study. 200 patients got full dose of steroids and they were assigned as either aggressive or expectant group.
- 62.5% patients were in the age group 20- 29 years
- 64.5% of severe preeclamptic women were primi
- Recurrent preeclampsia in 37.9% patients
- 8% patients had mother with preeclampsia
- 18% patients had sister with preeclampsia
- Mean systolic blood pressure was 157.4 mmHg
- Mean diastolic blood pressure was 105.2 mmHg

Likelihood of developing preeclampsia is increased according to

BJOG Oct 2003

- Primi.
- Age >30.
- Family history.
- BMI >35.

Sibai et al showed risk of developing preeclampsia in next pregnancy being 45.5% out of them 21% go for severe preeclampsia.

INDICATION FOR TERMINATION

	Study	maternal indication	fetal indication
a.	Blackwell SC[3] & others '02	80.0%	30.0%
b.	Hall DR, Odendaal HJ[15] '00	55.0%	45.0%
c.	Our study	66.0%	34.0%

Most common maternal indication being imminent eclampsia. Our study values are in between the both studies available.

MODE OF DELIVERY

Though mode of delivery had not shown to influence the fetal outcome, LSCS rate was higher in expectant group.

	Study	Vaginal	LSCS
a.	Hall BR & Odendaal HJ[15] 2000	18.5%	81.5%
b.	Nasser et al[23] 1998	48.3%	51.7%

c.	Murphy DJ, Stirrat M[22] 2000	20.0%	80.0%
d.	Railton A & Allen DG[28] 1987	25.0%	75.0%
e.	Our study	62.0%	38.0%

Our study is similar to Nasser study.

PROLONGATION OF PREGNANCY

	Study	**Mean prolongation of pregnancy**
a.	Odendaal HJ et al[25] 1990	7.1 days
b.	Sibai et al[36] 1994	15.4 days
c.	Vissur&wallenberg[41] 1995	14 days
d.	Yong, Li R[47]	11days (28-31weeks) 08days (32-33weeks)
e.	Railton A, Allen DG[28] 1987	11.4 days
f.	Olah KS Redman CW[26]	9.5 days
g.	Murphy DJ&Stirrat GM[22]2000	14 days
h.	Our study	7.54days

Our study similar to Odendaal HJ'study.

MATERNAL OUTCOME

Comparison of major complications in both the group.

	Study	**Complications**
a.	Odendaal et al[25] 1990	No increase in complication.
b.	Sibai et al[36] 1994	No increase in complication.
c.	Hall et al[15] 2000	No maternal death. 3 needed ICU. 1 needed dialysis.
d.	Haddad B Deis S[13] 2004	No maternal death or eclampsia. Morbidity similar in both groups.
e.	Railton A, Allen DG[28] 1987	23.2% had increase in major complications.
f.	Our study 2010	11.0% in aggressive 18.0% in expectant group. Statistically insignificant.

MATERNAL COMPLICATIONS

Study	Abruption	Pulmonary edema	HELLP	Eclampsia	renal failure
Hall and collegues[15] 2000	20%	2%	5%	1.2%	0.3%
Vissur& Wallenberg[41]1995	5%	--	--	1.9%	--
Murphy DJ&Stirrat GM[22] 2000	1.5%	--	21%	1.4%	1.3%
Olah KS Redman CW geeth[26] 93	--	--	14.2%	--	.5%
Our study	8%	2%	3%	2%	1%

Our study showed relatively low incidence of abruption and HELLP than Hall's study. No maternal death in our study.

- Among 3 patients complicated by HELLP/DIC, 2 patients had coagulopathy with prolonged clotting time bleeding from wound on table and they were treated with FFP and platelet transfusion.

Third patient who developed HELLP was terminated and treated with 8 units platelets.

- 1 patient who had compromised renal function was treated conservatively under nephrologist guidance
- 1patient developed postpartum eclampsia on 2nd post operative day and was started on magnesium sulphate.
- 2 patients developed pulmonary edema and were treated vigorously under anaesthetist supervision.

These situations explain the necessity of intensive care facilities in the management severe preeclampsia.Hemodynamic monitoring plays a major role in the treatment and trained persons and anaesthetist were available all the time for central vein catheterisation and monitoring.

PERINATAL OUTCOME

i) Perinatal mortality

	Study	**Perinatal loss**
a.	Railton A, Allen DG[28] 1987	24.5%
b.	Odendaal HJ[25] 2000	22.3%
c.	Hall DR[15] 2000	24.0%

d.	Murphy DJ & Stirrat GM[22] 2000	30.0%
e.	Haddad B, Dies S[13] 2004	10.7%
f.	Our study	36.0%

Our study is similar to Murphy's and is closer to 3 other studies.

ii. Birth Weight

	Study	**Aggressive**	**Expectant**
a.	Sibai et al[36] 1994	1.2kg	1.62kg
b.	Our study	1.33 kg	1.61kg

Our study correlated with Sibai et al and all others studies, showed higher birth weight by expectant management.

iii. Survival rate

	Study	**Aggressive**	**Expectant**
a.	Sibai et al[36] 94	24.0%	65.0%
b.	Hall DR[15] 2000	70.0%	94.0%
c.	Our study	48.6 %	73.5%

Railton A, Allen DG 1987 showed 100% Survival rate of babies born > 30 weeks in either group.

Our study showed higher Survival rate in babies of higher gestational age and birth weight and it was between both studies.

iv. Neonatal complications

	Study	**Hospital Stay(days)**	
		Aggressive	**Expectant**
a.	Sibai et al[36] 94	30.6	20
b.	Olah KS[26] 93	>15	7.4
c.	Our study	13.4	9.6

Our study showed expectant management babies had highest survival rate and lower neonatal complications. In this study admission was higher than that of Sibai et al because of lower birth weight but hospital stay as that of Olah due to advancement of neonatal care in the past 2 decades.

SUMMARY

In our study 100 patients with severe preeclampsia 28-32 weeks who were managed aggressively were compared with 100 patients with severe preeclampsia 28-32 weeks who were managed expectantly and following parameters were analysed.

- Majority of the patients around 62.5% belong to the age group between 20-29 years.
- Almost two third (62.5%) patients were primi.
- Mean gestational age on admission was 31 weeks.
- 37.9% patients had recurrent preeclampsia.
- 26% patients had family history of preeclampsia.
- Mean systolic blood pressure was 157.4mmHg and mean diastolic blood pressure was 105.2mmHg.
- Majority of the patients had normal fundus. Around one fifth (19%) had hypertensive retinopathy.
- Majority of the patients were given magnesium sulphate.

- Though major maternal complications were higher in expectant group it was proved to be statistically insignificant (P =0.15).
- 8 patients had abruption, 3 patients had DIC, 2 patients had eclampsia, 2 patients had pulmonary oedema and no maternal death.
- 66% patients were terminated for maternal indications and 44% patients were terminated for fetal indications.
- Though LSCS rate was higher in expectant group (38% vs 21%) but this was attributed to increased fetal salvagability and post caesarean pregnancy in expectant group.
- Mean prolongation of pregnancy was 7.54 days.
- Perinatal loss was significantly lower in expectant group 36% vs 63% proved to be statistically significant (P<0.001).
- Perinatal loss was not influenced by LSCS. Perinatal loss in LSCS was 57.0% and 36.8% in aggressive and expectant group.
- Perinatal loss was higher in the patients delivered <48 hours of steroid.
- Mean birth weight was higher in expectant group (1.33kg vs 1.58kg). It was found to be statistically significant (p<0.001) .

- Mean gestational age at delivery was higher in expectant group (31.64 vs 30.55 weeks). It was found to be statistically significant ($p<0.01$) .
- Expectant group had lower mean stay of hospitalisation(9.6 vs 13.4 days).
- Babies in expectant group had higher survival rate(73.5% vs 48.6%). It was found to be statistically significant ($p<0.001$) .

CONCLUSION

The expectant approach for management of severe preeclampsia remote from term results in better obstretic outcome in the form of

a. Lower perinatal mortality.

b. Higher perinatal & neonatal survival

c. Lower neonatal complications.

d. Higher birth weight.

e. Without increased maternal morbidity and mortality

The success rate of expectant management depends on both gestational age and maternal and fetal condition at the time of admission. Since maternal and perinatal complications are significantly increased in these patients, expectant management should be done only in well selected patients only at tertiary centres where adequate maternal and neonatal intensive care facilities are available.

Expectant management delays delivery and enhance fetal maturity and does not appear to be associated with increased risk of maternal morbidity and mortality

PROFORMA

Name : **Obstetric score:**

Age : **LMP :**

Address : **EDD :**

Ip. No : **Blood group:**

Occupation : **Ht :** **Wt:**

Booked/ not : **HIVstatus:**

Referred/not :

Complaints

Present h/o :

period of amenorrhea

pain abdomen

edema feet

vomiting

headache

oliguria

bluring of vision

palpitation

Present Pregnancy

I trimester

II trimester

III trimester

Past obstetric history

Medical history

Family history

General Examination :

Temperature	:	CVS	:
Pulse rate	:	RS	:
Blood pressure	:	CNS	:
Respiratory rate	:		
BMI	:		
Edema	:		
Anaemia	:		

Per abdomen :

Pervaginal :

Investigation

Urine - albumin:	:	Hb	:
sugar	:	Pcv	:
		Platelet	:
Blood - sugar	:	LFT	:
urea	:		
creatinine	:		
uricacid	:		
electrolyte	:		

Ultra sound :

Anti-hypertensive : Drug : dose :

Gravidogram

Date U/A Wt Sfh AG BP Immiment symptom

$Mgso_4$:

Time Dose Temp RR I/O DTR PERL U/A

Maternal complications :

Mode of induction :

Indication for termination : Maternal / fetal

Vaginal delivery / LSCS

Latency interval:

Intra/ post partum complications

Baby : alive / still birth

gestational age

birth weight

apgar score

admitted / not

Follow up : NICU stay

Neonatal complications

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ABBREVIATION

ACOG	-	American College of Obstetrics and Gynecology
ANP	-	Atrial natriuretic peptide
ARDS	-	Acute respiratory syndrome
BJOG	-	British Journal Obstetrics andGynecology
CTG	-	Cardiotocography
DIC	-	Disseminated intravascular coagulation
HCG	-	Human chorionic gonodotropin
HLA	-	Histocompatability antigen
IUGR	-	Intra uterine growth restriction
LFT	-	Liver function test
LSCS	-	Lower segment caesarean section
NICU	-	Neonatal intensive care unit
NST	-	Nonstress test
USG	-	Ultrasonography

MASTER CHART - AGGRESSIVE GROUP

S. No	Age	Parity	Past History	Family History	Blood Pressure (mmHg)	MGSO4	Fundus	Induction	Latency (Hours)	Mode of Delivery	Indication	Gestational Age (Weeks)	Birth Weight (KG)	Alive / Stillbirth	NICU stay (days)	Maternal Complications
1	19	E	B	B	142/112	A	A	A	70	A	-	32	1.7	A	10	-
2	22	B	B	B	154/90	B	A	B	53	B	A	31	1.27	A	A	-
3	25	A	-	A	158/102	A	A	B	73	A	-	29	1	A	A	-
4	20	A	-	B	170/92	A	A	A	82	A	-	31	1.2	A	A	A
5	27	A	-	B	172/120	A	C	A	52	B	E	32	1.38	B	-	-
6	17	B	B	B	144/104	A	A	A	86	A	-	30	1.24	A	A	-
7	23	C	A	B	160/94	A	A	B	68	A	-	32	1.78	A	11	-
8	24	B	B	B	172/96	A	A	A	90	A	-	29	1.1	B	-	-
9	18	A	-	B	142/106	A	B	A	78	A	-	28	1.16	B	-	-
10	26	A	-	A	152/122	A	B	A	54	B	D	31	1.45	A	13	-
11	20	A	-	B	166/108	A	A	A	80	A	-	32	2.1	A	4	-
12	25	B	A	B	164/118	A	A	B	87	A	-	31	1.24	A	16	-
13	19	A	-	B	146/110	A	A	A	60	A	-	31	1.3	B	-	B
14	22	A	-	B	156/98	A	A	A	85	A	-	29	1.15	A	A	-
15	31	A	-	B	176/100	A	A	A	67	B	B	31	1.22	A	A	-
16	21	C	B	A	148/114	A	A	A	89	A	-	30	1.25	A	15	-
17	32	B	B	B	168/102	A	A	B	65	A	-	32	1.62	B	9	-
18	23	A	-	B	150/98	A	A	B	77	A	-	32	1.58	A	A	-
19	17	A	-	B	140/108	A	A	A	49	A	-	29	1.18	B	-	-
20	33	D	A	B	174/92	A	A	A	56	A	-	31	1.42	A	17	-
21	17	C	B	B	150/120	A	A	B	53	B	A	32	1.28	B	-	-
22	22	B	B	B	142/110	A	B	A	78	A	-	31	1	A	A	-
23	21	A	-	A	160/94	A	A	A	86	A	-	31	1.2	A	A	-
24	25	B	A	B	148/114	A	C	A	68	A	-	30	1.24	B	-	-
25	19	A	-	B	174/90	A	A	B	87	A	-	32	1.7	A	7	-

S. No	Age	Parity	Past History	Family History	Blood Pressure (mmHg)	MGSO4	Fundus	Induction	Latency (Hours)	Mode of Delivery	Indication	Gestational Age (Weeks)	Birth Weight (KG)	Alive / Stillbirth	NICU stay (days)	Maternal Complications
26	22	A	-	B	172/108	A	A	A	70	B	D	31	1.65	A	8	-
27	26	A	-	B	158/98	A	A	A	79	A	-	32	1.4	A	A	-
28	18	B	B	B	144/106	A	A	A	54	A	-	29	1.12	B	-	-
29	20	D	A	B	152/94	B	A	B	90	A	-	33	1.8	A	4	-
30	23	A	-	B	162/90	A	A	A	92	A	-	32	1.48	A	12	-
31	27	A	-	B	168/126	A	B	A	59	B	E	28	1.18	B	-	A
32	32	A	-	A	148/96	B	A	A	65	A	-	32	1.45	A	9	-
33	16	A	-	B	176/116	A	A	A	91	A	-	31	1.2	A	17	-
34	22	B	B	B	164/104	A	A	B	85	A	-	31	1.55	A	11	-
35	26	A	-	B	154/92	A	A	A	50	A	-	28	1.12	A	A	-
36	20	C	B	B	146/102	B	A	A	89	A	-	31	1.34	A	14	-
37	25	A	-	B	162/118	A	B	A	64	A	-	29	1.2	B	-	-
38	24	A	-	B	156/106	A	A	A	75	A	-	32	1.63	A	A	-
39	19	A	-	B	176/108	A	A	A	52	B	D	28	1.26	B	-	-
40	30	B	A	B	140/100	B	A	B	58	A	-	31	1.23	A	A	B
41	20	B	B	B	140/96	B	A	A	52	A	-	32	1.33	A	A	-
42	25	A	-	B	160/116	A	B	A	58	B	B	29	1.24	A	A	-
43	19	A	-	B	176/102	A	A	A	64	A	-	30	1.2	B	-	-
44	26	C	A	B	162/90	B	A	A	66	A	-	32	1.64	A	6	-
45	27	A	-	A	144/106	A	A	B	58	A	-	30	1.2	A	18	C
46	22	A	-	B	164/94	A	A	A	79	A	-	30	1.4	A	17	-
47	30	B	B	B	172/108	A	A	B	83	B	A	31	1.25	A	16	-
48	19	D	A	B	168/104	A	A	A	85	A	-	30	1.25	A	19	-
49	21	A	-	B	158/96	A	A	A	50	A	-	32	1.76	A	A	-
50	16	A	-	B	146/118	A	B	A	91	A	-	28	1.21	B	-	-
51	22	A	-	B	154/110	A	A	A	83	A	-	31	1.28	B	-	-
52	32	A	-	B	166/98	A	A	A	68	A	-	30	1.25	A	20	A

S. No	Age	Parity	Past History	Family History	Blood Pressure (mmHg)	MGSO4	Fundus	Induction	Latency (Hours)	Mode of Delivery	Indication	Gestational Age (Weeks)	Birth Weight (KG)	Alive / Stillbirth	NICU stay (days)	Maternal Complications
53	23	B	B	A	148/108	A	A	B	76	A	-	29	1.08	B	-	-
54	20	A	-	B	170/120	A	A	A	71	B	D	32	1.68	A	A	-
55	26	A	-	B	142/106	A	B	A	85	A	-	31	1.5	A	16	-
56	25	A	-	B	174/100	A	A	A	69	A	-	29	1.12	A	A	-
57	33	C	A	B	152/114	A	C	B	92	B	B	32	1.72	A	8	-
58	17	B	B	B	156/104	B	A	A	76	A	-	31	1.22	A	A	-
59	24	A	-	B	144/90	B	A	B	70	A	-	31	1.46	A	17	-
60	29	A	-	A	174/98	A	A	A	70	B	C	30	1.12	A	A	-
61	18	D	B	B	144/112	A	B	A	58	A	-	31	1.38	A	17	-
62	24	B	B	B	172/94	A	A	A	79	A	-	32	1.53	A	A	-
63	21	A	-	B	154/102	A	A	A	80	A	-	32	1.47	B	-	A
64	22	A	-	A	140/92	B	A	A	62	A	-	31	1.26	A	B	-
65	26	A	-	B	148/104	A	A	A	82	A	-	29	1.8	A	A	-
66	22	C	A	B	156/100	A	A	B	56	A	-	32	1.64	A	7	-
67	25	A	-	B	152/114	A	A	A	88	B	D	28	0.9	A	A	-
68	24	B	B	B	148/108	A	A	A	49	A	-	29	0.85	B	-	-
69	16	A	-	B	174/94	A	A	B	53	A	-	31	1.22	A	A	-
70	20	A	-	B	150/110	A	A	A	69	A	-	28	0.95	B	-	-
71	22	A	-	B	160/124	A	B	A	87	B	D	32	1.9	A	16	-
72	26	A	-	B	162/128	A	A	A	48	A	-	31	1.28	A	A	-
73	27	C	A	A	146/98	A	A	B	77	A	-	33	2.2	A	4	-
74	23	B	B	B	176/118	A	A	A	70	B	D	32	1.44	A	11	-
75	25	B	A	B	164/106	B	A	B	74	B	B	29	0.98	A	A	-
76	35	A	-	B	144/96	A	A	A	50	A	-	32	1.72	A	5	E
77	20	A	-	A	166/98	A	A	B	78	A	-	28	0.9	A	A	-
78	32	A	-	B	178/120	A	A	A	84	A	-	28	0.75	B	-	-
79	23	B	B	B	142/92	A	B	A	52	A	-	31	1.3	A	A	-

S. No	Age	Parity	Past History	Family History	Blood Pressure (mmHg)	MGSO4	Fundus	Induction	Latency (Hours)	Mode of Delivery	Indication	Gestational Age (Weeks)	Birth Weight (KG)	Alive / Stillbirth	NICU stay (days)	Maternal Complications
80	27	C	A	B	140/116	A	A	A	64	A	-	31	1.35	A	-	-
81	28	A	-	A	144/110	A	B	A	77	B	D	29	0.82	A	-	-
82	29	B	B	B	172/102	A	A	A	58	A	-	30	1.55	A	-	-
83	30	A	-	B	160/120	A	C	A	83	A	-	28	0.78	B	-	C
84	31	A	-	B	158/92	A	A	A	86	A	-	32	1.3	B	-	-
85	33	A	-	B	146/104	A	A	B	51	A	-	28	0.9	A	A	-
86	34	B	A	A	156/98	A	A	B	70	A	-	33	1.82	A	18	-
87	28	A	-	B	142/114	A	B	A	86	A	-	29	0.85	A	A	-
88	33	A	-	B	174/106	A	A	A	52	A	-	28	0.8	B	-	-
89	32	B	B	A	160/94	A	A	A	49	A	-	30	0.95	A	A	-
90	40	A	-	B	170/90	A	A	A	63	A	-	32	1.69	A	11	-
91	28	A	-	B	140/98	A	A	B	73	A	-	31	1.18	A	A	-
92	38	A	-	B	176/112	A	A	A	88	B	B	32	1.5	A	8	-
93	30	B	B	A	166/108	A	A	A	56	B	A	31	1.41	A	13	C
94	34	A	-	B	158/100	A	A	A	91	A	-	30	1.25	A	21	-
95	33	A	-	B	140/104	B	A	A	64	A	-	32	1.52	A	A	-
96	29	B	A	B	172/118	A	B	B	78	B	E	30	1.27	A	20	-
97	32	A	-	B	148/96	A	A	A	53	A	-	31	1.08	A	A	A
98	28	C	B	B	154/110	A	A	A	51	A	-	31	1.26	B	-	-
99	28	A	-	-	152/112	A	B	A	64	A	-	31	1.2	A	A	-
100	26	A	-	-	160/114	A	B	A	58	A	-	31	1.22	A	A	-

MASTER CHART – EXPECTANT GROUP

S.No	Age	Parity	Past History	Family History	Blood Pressure (mm Hg)	MGSO4	Fundus	Induction	Latency Days	Mode of Delivery	Indication	Gestational Age (Weeks)	Brith Weight (KG)	Alive/ Stillbrith	NICU Stay (days)	Maternal Complications
1	19	A	-	B	172/112	A	B	A	21	B	C	33	2.15	A	3	-
2	24	B	A	B	154/110	A	A	A	8	A	-	34	1.72	A	10	-
3	35	B	B	B	158/90	B	A	B	5	B	B	33	1.77	A	11	-
4	25	A	-	A	144/108	B	A	A	3	B	B	32	1.27	A	A	-
5	33	B	B	B	154/114	A	B	A	7	A	-	31	1.56	A	12	-
6	22	A	-	B	140/98	B	A	A	4	A	-	28	0.85	B	-	-
7	26	A	-	B	176/106	A	A	A	3	A	-	32	1.50	A	9	A
8	16	A	-	B	142/120	A	B	B	4	B	D	31	1.45	A	18	-
9	24	A	-	B	156/94	A	A	A	4	A	-	31	1.51	A	A	-
10	34	B	A	B	148/96	B	A	B	6	A	-	33	1.62	A	10	-
11	31	A	-	B	172/116	A	A	A	4	A	-	31	1.40	A	16	D
12	21	A	-	B	160/102	A	A	B	23	B	D	34	2.20	A	4	-
13	27	A	-	B	164/100	A	A	A	4	B	C	28	1.10	A	17	-
14	36	D	A	B	146/104	B	A	A	12	A	-	33	1.76	A	A	-
15	20	A	-	B	168/118	A	A	A	3	A	-	30	1.26	B	-	E
16	28	A	-	B	174/106	A	A	B	9	B	D	34	1.75	A	15	B
17	18	A	-	B	164/94	A	A	A	8	A	-	34	1.78	A	5	-
18	23	A	-	B	160/102	A	A	A	20	B	B	33	2.10	A	2	-
19	32	B	B	A	144/124	B	B	B	3	B	A	29	1.20	A	A	-
20	33	B	B	B	162/112	A	A	A	8	B	A	33	2.18	A	4	-
21	23	A	-	B	156/110	A	A	B	17	B	E	31	1.26	B	-	A
22	25	A	-	A	174/98	A	A	A	8	A	B	34	2.00	A	2	-
23	20	A	-	B	142/108	B	A	A	4	A	-	29	0.850	B	-	-
24	17	A	-	B	178/114	A	A	B	3	B	D	30	1.50	A	17	-
25	24	A	-	B	140/90	B	A	A	6	A	-	33	1.58	A	A	-

S.No	Age	Parity	Past History	Family History	Blood Pressure (mm Hg)	MGSO4	Fundus	Induction	Latency Days	Mode of Delivery	Indication	Gestational Age (Weeks)	Brith Weight (KG)	Alive/ Stillbrith	NICU Stay (days)	Maternal Complications
26	28	A	-	B	154/100	A	A	A	18	B	B	34	2.10	A	02	B
27	34	C	A	B	144/120	A	B	B	10	A	-	32	1.62	A	12	-
28	27	A	-	B	166/106	A	A	A	11	A	-	33	1.78	A	9	-
29	21	A	-	B	158/94	B	A	A	12	A	-	32	1.62	A	15	-
30	18	A	-	B	176/104	A	A	B	4	B	D	34	1.36	A	A	-
31	29	B	A	B	146/116	A	A	A	5	A	-	33	1.70	A	16	-
32	30	B	B	A	162/102	A	A	B	4	A	-	28	1.00	B	-	E
33	26	A	-	B	170/126	A	B	A	9	A	-	34	1.86	A	8	-
34	37	C	A	B	144/106	B	A	A	3	B	E	30	1.12	B	-	A
35	20	A	-	B	154/96	B	A	A	5	B	B	31	1.24	A	A	-
36	31	B	B	B	152/108	A	A	B	3	A	-	33	1.65	A	14	-
37	19	A	-	B	176/92	A	A	A	18	A	-	34	2.20	A	3	-
38	22	A	-	B	140/118	A	B	A	7	A	-	31	1.20	A	A	-
39	31	B	B	B	168/92	A	A	A	10	A	-	32	1.59	A	A	-
40	24	A	-	B	176/114	A	B	B	11	B	D	33	1.88	A	9	-
41	27	A	-	B	140/108	B	A	A	4	A	-	31	1.26	B	-	A
42	29	A	-	B	142/90	B	A	A	9	A	-	34	1.64	A	10	-
43	18	A	-	B	164/112	A	A	B	19	B	D	30	2.20	A	2	-
44	23	A	-	B	146/110	A	A	A	10	A	-	34	1.70	A	A	-
45	30	B	A	B	180/120	A	B	B	8	B	A	34	1.49	A	15	-
46	20	A	-	B	158/106	A	A	A	6	B	B	30	1.25	A	14	F
47	39	B	B	A	170/94	A	A	B	9	A	-	33	1.90	A	4	-
48	22	A	-	B	160/104	A	A	A	5	A	-	31	1.47	A	17	A
49	33	C	B	B	140/116	A	A	A	8	A	-	34	2.15	A	2	-
50	28	B	B	B	154/102	A	A	B	3	B	D	29	1.08	A	A	-
51	18	A	-	B	156/96	B	A	A	12	A	-	33	1.66	A	8	-

S.No	Age	Parity	Past History	Family History	Blood Pressure (mm Hg)	MGSO4	Fundus	Induction	Latency Days	Mode of Delivery	Indication	Gestational Age (Weeks)	Brith Weight (KG)	Alive/ Stillbrith	NICU Stay (days)	Maternal Complications
52	26	A	-	B	178/102	A	A	A	4	A	-	30	1.24	A	13	C
53	21	A	-	B	142/98	B	A	A	11	B	-	34	1.98	A	4	-
54	25	A	-	A	152/118	A	B	A	7	B	B	29	1.48	A	10	-
55	19	A	-	B	144/104	B	A	A	3	A	-	28	.950	B	-	-
56	24	A	-	B	174/100	A	A	B	9	B	C	31	1.50	A	20	-
57	32	B	A	B	156/108	A	A	A	10	B	A	32	1.72	A	4	-
58	23	A	-	B	174/112	A	B	B	9	B	D	31	1.66	A	14	-
59	32	B	B	B	140/90	B	A	A	4	A	-	28	0.88	B	-	A
60	21	A	-	B	154/106	A	A	A	10	B	B	33	1.72	A	9	-
61	27	A	-	B	146/108	B	A	A	5	A	-	22	1.58	A	A	-
62	29	B	B	B	148/120	A	B	A	11	B	A	34	1.88	A	7	-
63	17	A	-	B	180/110	A	A	B	3	B	D	33	1.68	A	8	-
64	20	A	-	B	140/102	B	A	A	4	A	-	31	1.54	A	A	-
65	34	B	A	B	166/114	A	A	A	12	A	-	34	2.10	A	3	A
66	26	A	-	B	160/104	A	A	A	7	A	-	32	1.62	A	13	-
67	28	B	B	B	144/106	A	A	B	3	B	B	29	1.00	A	A	-
68	31	B	B	B	152/92	B	A	A	10	A	-	34	1.82	A	4	-
69	24	A	-	B	142/110	A	A	A	9	B	B	33	1.74	A	4	C
70	26	A	-	B	178/108	A	A	A	6	A	-	34	2.18	A	3	-
71	18	A	-	B	160/116	A	A	A	4	A	-	31	0.95	B	-	-
72	22	A	-	B	164/110	A	A	B	8	B	D	33	1.70	A	9	-
73	33	B	B	B	162/94	A	A	A	7	B	A	30	1.56	A	15	B
74	20	A	-	B	172/118	A	A	A	11	A	-	34	2.00	A	11	-
75	30	B	A	B	144/120	A	B	B	4	B	D	30	1.25	A	19	-
76	25	A	B	B	158/98	B	A	A	3	A	-	29	1.08	A	A	-
77	34	B	-	A	158/106	A	A	A	4	A	-	34	1.68	A	8	-

S.No	Age	Parity	Past History	Family History	Blood Pressure (mm Hg)	MGSO4	Fundus	Induction	Latency Days	Mode of Delivery	Indication	Gestational Age (Weeks)	Brith Weight (KG)	Alive/ Stillbrith	NICU Stay (days)	Maternal Complications
78	22	A	-	B	170/114	A	A	B	10	B	D	33	2.24	A	7	-
79	25	A	-	B	140/96	B	A	A	8	A	-	34	1.85	A	14	-
80	24	A	-	B	164/108	A	A	A	7	A	-	32	1.58	A	A	-
81	26	A	-	B	142/92	B	A	A	9	A	-	31	1.67	A	7	-
82	16	A	-	B	182/116	A	B	A	4	B	E	32	1.25	A	A	A
83	28	B	A	B	156/104	A	A	B	3	A	-	33	1.74	A	4	-
84	21	A	-	B	140/100	A	A	A	2	A	-	28	0.97	B	-	-
85	27	A	-	B	162/102	A	A	A	4	B	B	33	1.72	A	4	-
86	31	B	B	B	150/98	A	A	A	5	A	-	33	1.72	A	7	-
87	23	A	-	B	156/108	A	A	B	11	A	-	34	2.18	A	2	-
88	29	B	B	B	160/104	A	A	A	3	B	A	32	1.66	A	16	-
89	34	B	A	B	174/112	A	A	B	4	A	-	31	1.08	A	A	-
90	18	A	-	B	146/106	B	A	B	12	A	-	34	2.00	A	03	-
91	20	A	-	B	148/90	B	A	A	6	A	-	33	1.72	A	10	-
92	22	A	-	B	150/112	A	A	A	7	A	-	30	1.25	B	-	-
93	23	A	-	A	154/114	A	A	A	8	A	-	31	1.3	B	-	-
94	24	B	-	A	158/102	A	A	A	8	A	-	31	1.75	A	9	-
95	20	A	-	B	160/106	A	A	A	7	A	-	31	1.26	B	-	-
96	20	A	-	B	152/124	A	B	A	6	A	-	30	1.24	B	-	-
97	24	A	-	A	168102	A	A	A	9	A	-	31	1.4	B	-	-
98	25	A	-	B	156/108	A	A	A	10	A	-	31	1.6	B	-	-
99	27	B	B	A	160/124	A	B	A	14	A	-	32	1.8	B	-	-
100	28	A	-	B	156/110	A	B	A	12	A	-	32	2	A	8	-

KEY TO MASTER CHART

PARITY

A. Primi
B. Second gravida
C. Third gravida
D. Fouth gravida
E. Fifth gravida

PAST HISTORY

A. Past History Present
B. No past History

FAMILY HISTORY

A. Family History Present
B. No family history

$MgSO_4$

A. $MgSO_4$ given
B. $MgSO_4$ not given

FUNDUS

A. Normal
B. Grade I Hypertensive retinopathy
C. Grade II Hypertensive retinopathy

INDUCTION

A. Induced
B. Not induced

MODE OF DELIVERY

A. Vaginal
B. LSCS

INDICATION

A. Past LSCS
B. Non reassuring CTG
C. Mal Presentation
D. Failure to progress / unfavourable cervix
E. Abruption

NICU STAY

A. Neonatal death

MATERNAL COMPLICATIONS

A. Abruption
B. HELLP / DIC
C. Eclampsia
D. Renal failure
E. Pulmonary edema

ALIVE/STILL BIRTH

A. Alive
B. Still Birth